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CHOLECYSTITIS WITH PERFORATION

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IN AN attempt to determine the true incidence of perforation of the gallbladder in a large series of necropsies, a review was made of 12,000 consecutive routine autopsies done at the Los Angeles County Hospital from April 1936 to January 1942. It is evident from the results of this large series of necropsies that the incidence and mortality of perforation of the gallbladder are much higher than heretofore generally believed. The argument for early operation for acute cholecystitis as a means of lowering the mortality from perforation is further strengthened by the presentation of an analysis of 105 cases of acute cholecystitis in which operation was performed at the White Memorial Hospital, Los Angeles, in the period between 1924 and 1944 and at the Glendale Sanitarium and Hospital, Glendale, Calif., in the period between 1927 and 1944.

A vast amount of writing has been done on the subject of acute cholecystitis, but a comprehensive review of the literature covering the last decade indicates that debate is still going on regarding early or delayed operation. The trend in recent years, however, is definitely toward early surgical intervention. Failure of many physicians heretofore to accept early operation as the logical means of limiting the morbidity and lessening the mortality of acute cholecystitis seems to result from a failure to appreciate the incidence and gravity of perforation. Many, it would seem, have been content to risk this "supposedly" infrequent ominous development rather than subject the patient to the "alleged" dangers of an early operation.

We feel that the subject of perforation is of much practical importance, for on its frequency and inherent dangers should rest largely the decision as to the most opportune time to operate for acute cholecystitis. If it can be shown that the incidence and mortality are

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high then the brief for early operation is strengthened, and conversely, if perforation of the acutely diseased gallbladder is rare and the mortality low delay in surgical intervention would not be so potentially dangerous.

FREQUENCY OF PERFORATION

Considerable data are available in the literature on the incidence of perforation in cases of actual disease of the gallbladder. Cowley and Harkins (1943)[1] have made perhaps the most complete review of the literature on the incidence of perforation, extending from 1924 to 1942 and embracing the data presented by eighteen authors, including also a series of their own of 25 cases of perforated gallbladder. The average of total figures cited in a review of twelve thousand, nine hundred and fifteen operations on the gallbladder showed perforation occurring in 2.8 per cent of the cases. Focusing on acute conditions within the gallbladder, of which there were 2,261 cases, one finds that perforation occurred in 13 per cent. The mortality rate in cases of perforation was 20.8 per cent.

Heuer (1937)[2] concluded from his review of the literature that gangrene and perforation of the gallbladder occurred in 20 per cent of the cases of acute cholecystitis in which the pathologic process was not interrupted by surgical treatment, while his review of the experiences of fourteen authors, citing 500 cases of perforation, showed that the mortality varied between 15 and 65 per cent, averaging 45 per cent.

Perforation itself represents the end result of a progressive pathologic change in the acute condition of the gallbladder, the incidence and seriousness of which, it appears, have not been appreciated. If one judges from the content of earlier papers by Branch and Zollinger,[3] Smith,[4] Cave[5] and others, it would appear that it was felt that perforation was rather unusual or that if it did occur localization would generally be the rule.

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1. Cowley, L. L., and Harkins, H. N.: Perforation of the Gallbladder: A Study of Twenty-Five Consecutive Cases, Surg., Gynec. & Obst. **77**:661-668 (Dec.) 1943.

2. Heuer, G. J.: The Factors Leading to Death in Operations upon the Gall-Bladder and Bile-Ducts, Ann. Surg. **99**:881-892 (June) 1934; Surgical Aspects of Acute Cholecystitis, ibid. **105**:758-764 (May) 1937.

3. Branch, C. D., and Zollinger, R.: Acute Cholecystitis: A Study of Conservative Treatment, New England J. Med. **214**:1173-1177 (June 11) 1936.

4. Smith, M. K.: The Treatment of Acute Cholecystitis, Am. J. Surg. **40**: 192-196 (April) 1938.

5. Cave, H. W.: Immediate or Delayed Treatment of Acute Cholecystitis: Liver Shock and Death, Surg., Gynec. & Obst. **66**:308-314 (Feb.) 1938.

Contrary to these, Sanders,[6] Heuer,[2] Hotz,[7] Stone[8] and their colleagues found that perforation without localization was by no means infrequent.

With respect to the appendix, Heuer (1937)[2] cited the incidence of gangrene and perforation in acute appendicitis to be 17.5 per cent in a series of 593 cases as compared with an incidence of gangrene and perforation of 15.7 per cent in a series of acute conditions within the gallbladder observed during the same period, which fact should dispel the contention that acute perforation of the gallbladder is rare when compared with that of acute perforation of the appendix. When this comparison is made, it is interesting to note that Hotz (1939)[7] concluded from his investigations that the incidence of peritonitis associated with perforation of the gallbladder was found to rank as high as that of peritonitis associated with the acute perforation of the appendix, which was reported by McClure and Altemeier,[9] as occurring in 53 per cent of their series of cases.

The surgical significance of this comparison should lead to the definite conclusion that just as surely as the mortality and morbidity rates have been lowered by early operation for acute appendicitis just so must they be lowered by operation for the acutely diseased gallbladder, by an interruption at an early stage of the pathologic process which will so frequently result in the catastrophe of perforation, with its high incidence of fatality.

ANALYSIS OF CASES OF PERFORATION

Considerable data appear in the literature on the incidence of perforation in gallbladder disease, but little, comparatively speaking, is found regarding the incidence of perforation of the gallbladder as a cause of death. Our series reviews 12,000 consecutive routine necropsies performed at the Los Angeles County Hospital from April 1936 to January 1942 and reveals 32 instances in which perforated gallbladder was found to be the cause of death, or an incidence of 0.26 per cent. In other words, approximately 1 in every 375 persons succumbed to a perforated gallbladder, surely a much higher incidence than generally heretofore believed.

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6. Sanders, R. L.: Perforation of Gallbladder: Analysis of Forty-Six Cases, Surgery **1**:949-958 (June) 1937.

7. Hotz, R.: Perforated Cholecystitis, Am. J. Surg. **44**:706-711 (June) 1939; Acute Cholecystitis with Discussion of Factors in Morbidity and Mortality, ibid. **44**:695-705 (June) 1939.

8. Stone, W. W.: Perforation of the Gallbladder Occurring in the Late Stage of Pregnancy, J. A. M. A. **109**:1903-1904 (Dec. 4) 1937.

9. McClure, R. D., and Altemeier, W. A.: Acute Perforated Appendicitis with Peritonitis, Ann. Surg. **105**:800-814 (May) 1937.

For instance, Karullon, cited by Nix (1940),[10] reported 6,114 consecutive necropsies in only 3 of which death was due to perforation of the gallbladder, whereas our series, showing 1 death due to perforation in every 375 deaths from all causes, should surely dispel the contention that perforation is infrequent, rare or seldom fatal!

Sex.—Most series show all types of gallbladder disease to occur with greater incidence in women; however, in this series of cases of perforation, owing to some unknown vagary, 22, or 68 per cent, of the patients were men, while 10, or 31 per cent, were women, a sex ratio quite the reverse of that usually found. A ratio favoring the men occurred in Sanders' (1937)[6] series of 46 perforations of the gallbladder in 22 women and 24 men.

Age.—Compilation of table 1 revealed the youngest patient to be 42 years and the oldest 91 years old. The literature shows that by far the majority of perforated gallbladders occur in patients past 50 years. Schaeffer (1942)[11] gave the average age of his 20 patients to be 59 years. In our series, 91 per cent of the patients were past 50 years.

Duration of Present Illness.—In the present autopsy series of 32 cases, in 16, or 50 per cent, the onset of the present attack to the time

TABLE 1.—*Age Incidence of Patients with Perforation of the Gallbladder*

Age, Years	Number of Cases	Percentage
40-49	3	8.1
50-59	7	21.8
60-69	7	21.8
70-79	10	31.2
80-89	4	12.5
90-99	1	3.1

of hospitalization did not exceed seven days, while the remainder were of varying or unstated time. However, many of the patients in the former group presented acute pathologic conditions. If they had not been conservatively treated, the condition undiagnosed or misdiagnosed or if early instead of delayed operation had been done, it is felt that a considerable number of lives would have been saved.

Classification as to Type of Perforation.—Table 2 compares the incidence of free and localized types of perforations as occurring in our series with the incidence of those tabulated from the literature. Free perforation of the gallbladder with generalized peritonitis (type I of Niemeier) occurred in 42 per cent of our cases, as shown in table 3. This is considerably higher than the incidence found by Cowley and

10. Nix, J. T., in discussion on McCloskey, J. F., and Lehman, J. A.: Surgical Management of Acute Cholecystitis, Rev. Gastroenterology **7**:176-183 (March-April) 1940.

11. Schaeffer, R. L.: Acute and Chronic Perforations of the Gallbladder, Pennsylvania M. J. **45**:566-569 (March) 1942.

Harkins (1943),[1] who reported a series of 25 cases, with an incidence of free perforation in 24 per cent. Hotz (1939),[7] however, in reporting a series of 574 cases of acute conditions of the gallbladder, found 69 instances of perforation, or an incidence of 12 per cent. Peritonitis was present in 77 per cent of the patients at the time of operation, and 60 per cent of these presented diffuse or general peritonitis. A bile peritonitis was described in 6 cases, or 17 per cent of our series. These were not confused with a bile-tinged ascitic fluid as found in jaundice, so far as could be determined from available data.

TABLE 2.—*Incidence of Free and Localized Perforations of the Gallbladder*

Author	Year	Cases of Free Perforations		Cases of Perforations with Localization	
		Number	Percentage	Number	Percentage
Niemeier, O. W.: Ann. Surg. **99**: 922 (June) 1934.	1934	2	25.0	6	75.0
Sanders [6]	1937	4	8.6	42	91.0
Pennoyer, G. P.: Ann. Surg. **107**: 543 (April) 1938	1938	7	....	..	....
Stone, W. W., and Douglas, F. M.: Am. J. Surg. **45**: 301 (Aug.) 1939.	1939	6	35.0	11	64.7
Hotz [7]	1939	53	76.7	16	23.1
Atlee, J. L., and Atlee, J. L., Jr.: Pennsylvania M. J. **44**: 731 (March) 1941.	1940	4	26.6	11	73.3
Glenn, F., and Moore, S. W.: Arch. Surg. **44**: 677 (April) 1942	1942	3	12.0	22	88.0
Schaeffer [11]	1942	14	70.0	6	30.0
Cowley and Harkins [1]	1943	6	24.0	19	76.0
Johnstone and Ostendorph *	1944	14	42.4	19	57.6
Total or average		113		152	

* Total cases of perforated gallbladder = 32. Of these, there were 14 cases of free perforation of the gallbladder with generalized peritonitis and 19 cases of localized perforation, 1 case revealing both a free and a localized perforation.

TABLE 3.—*Classification of Series of Perforations According to Niemeier*

		Number of Cases	Percentage
Type I.	Acute free perforation with generalized peritonitis	14	42.4
	("Bile peritonitis" 6)		
Type II.	Pericholecystic abscess with localized peritonitis (subacute)	14	42.4
Type III.	Biliary fistula (chronic)	5	15.1
	Into duodenum 4		
	Into liver * 1		
Total cases		33 *	

* Total cases = 32. One case, counted twice, had both a free perforation of the gallbladder, with generalized peritonitis, and a perforation into the liver.

It is interesting to note that of the 4 cases of perforation into the duodenum 2 of these also revealed at necropsy a generalized peritonitis due apparently to subsequent perforation of the jejunum in 1 instance and to leakage from an unstated site in the other.

One death, not included in the series of perforated gallbladder, was stated by the surgeon performing the autopsy as being due to acute cholecystitis, cholelithiasis and perforation of the common duct, with

localized peritonitis. Why the perforation was in the common duct rather than in the gallbladder was not clear.

Incidence of Gallstones.—Stones were found to be present in 84 per cent of these cases and were not found in 4 cases, or 12 per cent. One instance of spontaneous cholecystoduodenostomy, resulting from extrusion of gallstone into the duodenum, resulted in intestinal obstruction and perforation of the jejunum, with consequent generalized peritonitis. Schaeffer[11] (1942) reported all his 20 patients with perforation, with the possible exception of 1, as having stones.

Diagnosis and Disposition of Cases.—As shown in table 4, the 32 cases comprising our series were divided into three groups. It is striking to note that group III, representing the cases in which no diagnosis had been made prior to coming to necropsy, numbered 16, which is 50 per cent of the total series. Touroff (1934)[12] found in a review of 429 cases of cholecystitis that minimal or absent clinical manifestations

TABLE 4.—*Diagnosis and Disposition of Cases of Perforated Gallbladder*

	Cases of Conservative Treatment (Medically)		Cases of Delayed Operation	
	Number	Percentage	Number	Percentage
Group I. Pathologic changes in gallbladder diagnosed on patient's admission to hospital	6	17.6	2 (cholecystostomies)*	8.8
Group II. Suspected pathologic changes in gallbladder diagnosed on admission to hospital	4 (aspiration of mass in 1)	11.7	2 (exploratory laparotomies)†	5.5
Group III. Condition undiagnosed prior to necropsy	15 (peritoneoscopy in 1)	47.0	2 (exploratory laparotomies)†	5.5
Group IV. Antemortem diagnosis unstated.....	1 case	2.8		

* Definitive surgical treatment (i. e., on the gallbladder).
† Other surgical treatment.

existed in 75 cases of pathologically proved acute cholecystitis. This may partially explain the failure of diagnosis in a large number of the cases in our series, for in the elderly patients, or those past 50 years, in which the incidence and mortality of perforation are greatest, the clinical manifestations are often minimal or even absent. The majority presented an acute exacerbation of a chronic cholecystitis, with a few patients with acute cholecystitis, a considerable number of whom we feel would not have succumbed to perforation had they not been treated medically or conservatively, as were 26, or 81 per cent.

The majority of the cases, as evidenced by the incidence of gallbladder calculi in 84 per cent, represented biliary disease of long stand-

12. Touroff, A. S. W.: Acute Cholecystitis: A Study of Seventy-Five Proven Cases with Subsiding or Subsided Clinical Manifestations at the Time of Operation, Ann. Surg. **99**:900-913 (June) 1934.

ing. If in these cases there had been an interval cholecystectomy early in the course of the disease, the progressive and irreversible pathologic state, ending with gangrene and perforation, would not have developed.

In 2 cases, delayed operation was instituted, cholecystostomy or simple drainage of an abscess being employed. These 2 cases, or only 6.2 per cent, represent the only definitive surgical treatment done on the gallbladder itself. In 4 other cases, surgical treatment other than definitive operation on the gallbladder was done. In this group are found 4 patients on whom exploratory laparotomies were performed. One was operated on for possible intestinal obstruction, but a perforated gallbladder proved to be the unrecognized pathologic change. (In this case the diagnosis had been acute cholecystitis four months previously, and the patient had been treated conservatively.) The second laparotomy was done for possible intestinal obstruction or acute appendicitis, and a postoperative diagnosis of diverticulitis of the sigmoid was made; the gallbladder was apparently not adequately explored to identify or do anything about the real pathologic changes. The third laparotomy was performed for a suspected perforated peptic ulcer, but instead a perforated gallbladder was found. Operation had been delayed eight days in this case, and the patient was being treated conservatively, even though gallbladder disease had been suspected when the patient was admitted to the hospital. The fourth laparotomy was done after a preoperative diagnosis of acute appendicitis. The postoperative diagnosis was the same plus acute cholecystitis, but, owing to the poor condition of the patient, the gallbladder was not dealt with. A delay of twenty-four hours here may have forfeited this patient's chance to survive, which he might have had if early cholecystectomy had been done.

REPORT OF A CASE

A case typical of others in the series review is here presented, illustrating the dangers and inadequacy of a medical or so-called conservative regimén in the presence of acute progressive disease of the gallbladder. It also serves to illustrate the need of educating the lay public as to the potential dangers of chronic cholecystic disease, especially that associated with gallstones, inasmuch as it is generally accepted that the pathologic basis of a perforation of the gallbladder is due in practically every instance to an obstruction of the cystic duct, usually by a calculus, and thus are initiated the ensuing progressive pathologic changes culminating in perforation.

W. M., an elderly, white, obese woman of 69 years, was admitted to the hospital Jan. 3, 1940 and died on January 7. The diagnosis when she was admitted was probable recurring cholecystitis. The subsequent history, obtained from a son, revealed that the patient had gallstones for many years, with episodes of nausea, vomiting and abdominal pain. The present attack began two days before hos-

pitalization and was subsequently followed by severe fever, chills and sweats, with the patient in a stupor for two days.

Physical examination was done by the intern. The temperature was 102.2 F. per rectum. The abdomen was relaxed except for voluntary muscle guarding; no mass was palpable. The patient complained on pressure anywhere, even on the chest. The white blood cell count was 15,700. The intern's diagnosis was impending uremia, chronic cervicitis and fever, of undetermined origin.

On January 3 the blood amylase was 156. The resident physician's impression was uremia. The causation was undetermined.

On January 6 the resident physician made a note regarding the history obtained, which revealed that the patient had been told for many years that she had gallstones and noted that the present attack may have had such a basis but that the definite diagnosis was not made as yet. On January 6 it was noted that the patient had evident jaundice. On January 7 a note from the resident physician read, "Patient died this morning. Cause of death not known." The clinical diagnosis as to the cause of death was hepatic and renal insufficiency secondary to recurrent cholecystitis.

At autopsy these observations were made: The abdominal cavity contained an estimated 3 to 4 cc. of foul-smelling, dark gray, semipurulent fluid. The appendix was retrocecal and was free from any inflammation. The gallbladder contained about one hundred small, faceted stones. The entire mucosa was extremely necrotic. The gallbladder was involved in a mass of omentum and fibrous tissue, which bound it firmly to the under surface of the liver and obscured all relations. The wall was greatly thickened and fibrotic, and there seemed to be a perforation on the anterior surface which led into a walled-off abscess, also containing several stones. The abscess in turn had penetrated into the duodenum through an opening measuring about 3 mm. in diameter. Also, the same abscess had penetrated into the peritoneum through another opening measuring about 3 mm. in diameter. The posterior wall of the gallbladder was extremely necrotic and lay in direct apposition with a large necrotic abscess in the liver, with which it seemed to communicate.

At microscopic examination of the gallbladder, the wall was extremely thick. There was perivascular round cell infiltration. The inner surface showed pronounced polymorphonuclear infiltration, and no epithelium was seen. Exudate from the hepatic abscess contained anhemolytic streptococci, and exudate from the gallbladder contained anhemolytic streptococci.

At autopsy the cause of death was established as chronic cholecystitis, cholelithiasis with perforation, abscesses of liver, acute peritonitis and bronchopneumonia.

Remarks.—This was a 69 year old woman treated for five days in the hospital, without a definite clinical diagnosis. She died as a result of a perforated gangrenous gallbladder, but if she had been treated surgically, rather than medically or conservatively, at an early date she might have lived.

ANALYSIS OF CASES OF ACUTE CHOLECYSTITIS IN WHICH OPERATION WAS PERFORMED

A series of 105 operative cases of acute cholecystitis is next presented in support of the view held by us that early operation, performed within

forty-eight hours of the onset of an acute attack of cholecystitis, is the logical and an effective means of reducing the mortality in this disease by interruption of the progressive process, which so often may result in the catastrophe of perforation. Moreover, cholecystectomy is felt to be the operation of choice, for when it is employed the pathologic condition is removed, thereby obviating the hazards of a later operation, as is often necessitated by cholecystostomy. Early operation increases the practicability of performing cholecystectomy, for within forty-eight hours the dense adhesions encountered in delayed operation are not yet established and the planes of dissection due to pericystic edema are easily defined.

A series of 56 consecutive cases of acute cholecystitis in which operations were performed at the White Memorial Hospital, Los Angeles, as were available from hospital records between 1924 and 1944, combined with a similar series of 49 cases from the records of the Glendale Sanitarium and Hospital, Glendale, Calif., from 1927 to 1944 (105 in all) were used for this study.

TABLE 5.—*Age Incidence in Operative Cases of Acute Cholecystitis*

Age, Years	Number of Cases	Percentage
30-39	11	10.4
40-49	23	21.9
50-59	34	32.3
60-69	18	17.1
70 +	17	16.1
Unstated	2	1.9

Age and Sex.—Of this series, 65 per cent of the patients were past 50 years, and, as seen from table 5, the age group from 50 to 59 years provided 32 per cent of the total. The sex ratio favored the women, and this has been true in most series reported in the literature, again an indication that disease of the gallbladder is commoner in women. However, our autopsy series showed the reverse of this, a vagary for which we have no explanation.

Duration of Acute Illness.—The duration of the acute attack or illness, i. e., from its onset to the time of operation, is shown in table 6, the cases being classified into three groups, according to interval of time.

Gangrene and Perforation.—In our series of 105 cases, gangrene occurred in 13, or 12 per cent. There was 1 death in this group, or a mortality of 7.6 per cent in gangrenous gallbladder. Perforation occurred only three times, with no deaths.

Type of Operation.—Referring to table 6, one will note that cholecystectomy was performed in 86, or 82.8 per cent, of the cases and cholecystostomy in 18, or 17 per cent. It is felt, from our experience,

that cholecystectomy is practically always feasible from both a technical and a clinical standpoint when the patient is operated on within forty-eight hours of onset.

Mortality.—The mortality rate for the 105 cases is 8.5 per cent. The mortality for the group in which operation was performed within forty-eight hours is 5.8 per cent, but if allowance is made for the surgical accident a rate of 2.9 per cent obtains. For patients operated on in the period forty-eight hours to seven days, the rate is 8.3 per cent. These figures are at variance with the findings of Bonn and Bachhuber [13] with regard to a similar interval. They found a mortality rate of 19 per cent for patients operated on within forty-eight hours of the onset in a series of 16 cases and a mortality rate for the group operated on in a forty-eight hour to seven day interval of only 7.8 per cent in 64 cases. For comparison with the series of Bonn and

TABLE 6.—*Duration of Present Attack, Type of Operation and Mortality*

Group	Time Interval	Number of Cases	Cholecystectomy	Cholecystostomy	Deaths	
					Number	Percentage
I	0-48 hr.	34	25	8	2*	5.8
II	2-7 days	36	30	6	3	8.3
III	7+ days	35	31	4	4	11.4
Total		105	86	18	9	8.5

* One death was due to a surgical accident, and the patient died on the table, with the operation uncompleted. Allowance for this case makes for a mortality in the forty-eight hour group of 2.9 per cent.

Bachhuber,[13] our lowest mortality is in the forty-eight hour group, which represents practically the same number of cases as in each of our other two groups. It is to be noted that there is a progressive increase of mortality proportionate to the time in delay of surgical intervention.

Our series of operative cases does not represent the work of one or two surgical teams but a series performed by a considerable number of different operators, not all of whom, by any means, were surgical specialists, the number also including general practitioners and occasional surgeons. This undoubtedly raised our mortality figures, but despite this our findings indicate the advisability and safety of early cholecystectomy.

Of the 2 patients who died in the forty-eight hour group, 1 died because of a surgical accident, the patient dying on the table ten minutes after operation was begun, and the other was a bedridden patient, under treatment for beriberi, who had a cardiac condition which

13. Bonn, H. K., and Bachhuber, C. A.: The Surgical Treatment of Acute Cholecystitis, Am. J. Surg. **49**:447-453 (Sept.) 1940.

would have made operation inadvisable at any time. This patient died fifteen hours following operation, from surgical shock and pulmonary edema. The associated pathologic change plus inadequate preoperative preparation, we feel, explains this second fatality and illustrates the fact that there are always a small group of cases of disease due to associated pathologic changes in which the patients should never be subjected to surgical treatment in any circumstances. Here, as in all cases, individualization is necessary.

Our mortality is lowest in the forty-eight hour group, of 34 cases, or a rate of 2.9 per cent, which compares favorably with the series reported by H. F Graham and M. E. Hoeffle[14] (1938) of 167 cases of acute cholecystitis, with a mortality of 3.5 per cent in patients subjected to operation within forty-eight hours.

SUMMARY AND CONCLUSIONS

1. There were found 32 instances of perforation of the gallbladder in a review of 12,000 consecutive routine necropsies done at the Los Angeles County Hospital from April 1936 to January 1942, giving an incidence of 0.26 per cent, or, in other words, in approximately 1 of every 375 cases coming to necropsy death was from a perforated gallbladder. Perforation of the gallbladder as a cause of death has heretofore been given little study in the literature, and these findings should dispel the contention that perforation is infrequent, rare or seldom fatal. Its true incidence places the acutely diseased gallbladder high on the list of potentially fatal conditions as found in acute conditions within the abdomen.

2. The inherent danger of peritonitis following perforation is a real one, and our group of 14 cases of free perforations with acute generalized peritonitis (Niemeier's type I) and 19 of localized perforation indicates that the perforation was not "walled off" in 43 per cent of the cases. This disproves the belief that free perforation is infrequent or that if it occurs it is generally "walled off."

3. The dangers of so-called medical, or conservative, treatment of acute cholecystitis are again emphasized, in that in 49 per cent of the total number of cases the conditions went on to perforation and death, being unrecognized and undiagnosed until necropsy.

4. Once the diagnosis of acute cholecystitis is made, particularly in elderly patients (past 50 years), surgical intervention as early as is consistent with adequate preparation of the patient and operating facilities should take place.

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14. Graham, H. F., and Hoeffle, M. E.: Acute Cholecystitis: Results of Operation Within Forty-Eight Hours of Onset of Symptoms, Ann. Surg. **108**: 874-876 (Nov.) 1938.

5. Interval cholecystectomy should be done in cases of recurrent chronic cholecystitis, thereby removing the possibility and dangers of progressive disease.

6. Early operation within forty-eight hours, employing cholecystectomy, is regarded as the logical and effective means of lowering the mortality in acute cholecystitis, the importance of adequate preoperative preparation and evaluation of the operative risk always being remembered.

7. A mortality rate of 2.9 per cent for patients operated on within forty-eight hours compares favorably with the findings of other proponents of early operation and leads to the conclusion that early surgical intervention will lower the mortality from acute perforated cholecystitis just as surely as it has lowered the mortality from acute perforative appendicitis, i. e., by removing the pathologic process before it has progressed to the stage of gangrene and perforation. The findings in 105 cases of acute cholecystitis in which operation was performed show that mortality increases in proportion to the time of operative delay.

REMOVAL OF SHELL FRAGMENT FROM LEFT VENTRICLE OF THE HEART

Report of a Case

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THE presence of a foreign body in the myocardium creates a dramatic and challenging situation. If the immediate needs of operation, such as cardiac tamponade, pericarditis or clotted hemothorax, are not present to clearly define surgical intervention, the question of surgical treatment is likely to harass the decison of the surgeon. Intracardiac foreign bodies have not been sufficiently frequent to establish a clear set of rules. As a result, the proverbial two schools of thought have not as yet merged. However, to date, opinion seems to favor removal or attempt at removal. Beck [1] urged the extraction of foreign bodies from the pericardial cavity or myocardium as early as possible and stated that the lapse of time does not mitigate against surgical intervention, even though clinically the patient shows improvement. (The improvement will invariably also be reflected in serial electrocardiograms.) While it is true that a foreign body in the cardiac muscle can become fixed by fibrous tissue, exploration is nevertheless recommended. Davies and Coope [2] held to the same opinion. In cases in which cardiorrhaphy for compression is indicated, they advised removal of the foreign body if it lies superficially and further recommended that "deeply embedded foreign bodies and those in the cavity of the heart are best left until later." A. Tudor Edwards [3] included in his indications for cardiac operation "the presence of foreign bodies, more particularly irregular shell fragments, impacted in the pericardium, in the walls of the heart, or within cavities of the heart."

The dangers of permitting a foreign body to remain in the musculature of the heart are threefold: (1) cardiac rupture, (2) migration into the adjacent cavity, with embolus formation or interference with the

1. Beck, C. S.: Surgery of the Heart and Pericardium, in Christopher, F.: A Textbook of Surgery, Philadelphia, W. B. Saunders Company, 1942, p. 1014.

2. Davies, M. H., and Coope, R.: War Injuries of the Chest, Edinburgh, E. & S. Livingstone, 1942, p. 103.

3. Edwards, A. T.: Wounds of the Thorax, in Bailey, H.: Surgery of Modern Warfare, ed. 2, Baltimore, Williams & Wilkins Company, 1942, vol. 1, p. 385.

capacity or function of the chamber, and (3) injury to a coronary vessel.

There are physicians, however, who would take a more conservative view. Bland [4] reported 8 cases of foreign bodies in and about the heart that were not removed. An analysis of these cases, however, affords no definite conclusions, for a sufficiently long enough time has not elapsed for the complete evaluation of the cases. In 1 instance of a bullet lodged within the right ventricle, the patient was experiencing substernal pain five months after the injury, with a sense of pressure radiating to the right upper arm, lasting for one to two hours and unaffected by motion, position or respiration. In another case, after an unsuccessful search for a foreign body in the left ventricle was made, the patient was still experiencing dyspnea on effort six weeks following injury. In all cases, however, the fact that the foreign bodies were permitted to remain, without immediately endangering the lives of the patients, does not serve to prove that such a course is the general procedure of choice and that later sequelae will not ensue.

The management of intracardiac foreign bodies will finally rest on principles evolved from a statistical analysis of the cases in which the patients were operated on and those in which the foreign bodies were left embedded. The present case report is offered as one to be considered for the ultimate over-all study.

REPORT OF A CASE

On the morning of June 24, 1944, just outside of Cherbourg, France, a 23 year old American soldier was lying in the prone position, waiting for the signal to move ahead. The signal was given, and the soldier was in the process of getting up when a mortar fragment stung him through his back. He continued to walk, however. He traversed 100 yards (274.32 meters) before he began to feel dizzy; then he experienced transient blindness, which, he believed, persisted for five minutes. He fell to the ground and became extremely dyspneic. Within fifteen minutes he was discovered by a company aid man and was started back through the chain of medical evacuation, receiving plasma, sulfadiazine, tetanus antitoxin, whole blood transfusions and penicillin in the advanced units. At the field hospital the same day, his respiratory distress was relieved by aspiration of 1,580 cc. of whole blood from the left side of the chest. The following day a good deal of mucus was aspirated from the trachea through an endotracheal tube. Tap of the chest at this time yielded only 20 cc. of blood. He was given another transfusion and kept under observation. Combined penicillin and sulfadiazine therapy was maintained. Still another attempt at aspiration, the ensuing day, proved fruitless.

When the patient was first seen by our department, he presented the appearance of a well built person who was cyanotic, slightly dyspneic and ill looking. He had been coughing up some old and fresh blood for the past few days and had begun to have a septic temperature reaching 102 F. He complained of attacks of vertigo

4. Bland, E. F.: Foreign Bodies in and About the Heart, Am. Heart J. **27**:588 (April) 1944.

and inability to breathe freely. Blood pressure prior to surgical treatment was 126 systolic and 60 diastolic. Examination of the chest revealed a wound of entrance at the left seventh interspace, paravertebrally (fig. 12), no wound of exit and the classic signs of fluid in the chest, occupying practically the entire

Fig. 1.—Roentgenogram taken July 21, 1944. In this view the foreign body is obscured by a massive left-sided hemothorax.

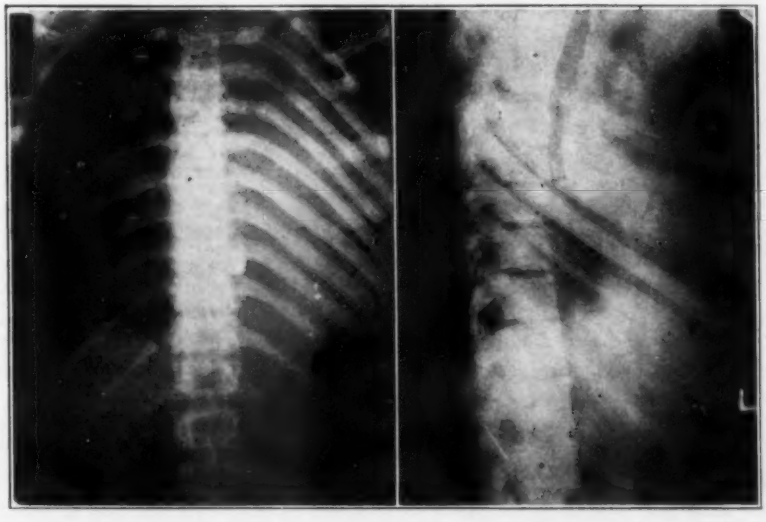

Fig. 2.—Overpenetrated roentgenograms taken July 21, 1944, showing the metallic fragment.

left side of the thorax. Thoracentesis secured only a few cubic centimeters of clotted blood, a culture of which was negative. A diagnosis of clotted hemothorax was made and was supported by roentgenologic observations (fig. 1).

The roentgenograms presented some interesting features. Inasmuch as the initial roentgenograms showed no foreign body because of overshadowing by the hemothorax (fig. 1), overpenetrated plates were made and they revealed a foreign body in the middle of the mediastinum in relationship to the fifth rib anteriorly, about 2.5 cm. to the left of the midline (fig. 2). Capt. M. Rakofsky, Chief of X-Ray Service, further stated that fluorscopic study seemed to indicate that the foreign body had cardiac motion. This, at the time, however, seemed illogical, inasmuch as the shell fragment had entered from the back and in a line that would have had to traverse the posterior wall as well as the chamber of the left ventricle in order to reach the apical position it now occupied. It appeared that the shell fragment was lodged in an inactivated portion of the lung, adjacent to the apex of the heart, and hence was receiving its motion by transmission. It was only after operation that it became evident how uniquely the fragment had ricocheted to its terminal position. Moreover, on retrospect, it is noteworthy that the blurred appearance of the foreign body (fig. 2) is in distinct contrast to the well defined edges of foreign bodies lodged in pulmonary or muscle tissue, when the roentgenograms are taken during inspiration. This feature is made particular notice of inasmuch as it might prove of diagnostic value in future considerations.

On July 27, 1944, with the patient under endotracheal anesthesia, operation was done, primarily to evacuate the left side of the thorax of clotted blood and secondarily to remove the foreign body. We were assisted by Major Frederick Steele. A transverse incision was made over the anterior aspect of the fifth rib, a portion of which was resected. Later, in order to provide greater exposure, the fourth left costocartilage was cut.

When the thoracic cavity was opened, an enormous amount of clotted blood and large pieces of well organized blood clot as well as sanguinous and serosanguinous fluid were removed. This procedure required a good deal of time. After its completion, exploration was carried out. The diaphragm was found to be elevated to the fifth rib and was covered with a fibrinous exudate about 4 mm. thick; the entire pleura, visceral and parietal, was similarly affected. Both lobes of the lung were completely collapsed, and, because the adherent, membranous exudate enveloped the heart and lungs in a continuous fashion, it was impossible to distinguish the one organ from the other.

After decortication of the lung, a cleavage line was established between the lung and heart and complete separation effected. An examination of the lung at this time failed to reveal the presence of a foreign body. It became apparent that the shell fragment, entering posteriorly, had cut through the lung, had struck and reflected from the anterior thoracic wall and had penetrated the heart. The site of entrance, the hemothorax, the burn marks of the missile in the lung tissue and a searing of the cardiac pleura gave evidence of this.

Although the prime purpose of the operation had been effected, it was deemed advisable to search for the foreign body. The condition of the patient was satisfactory. Secondary intervention would be complicated by the formation of adhesions, would be permissible only after a long convalescence and, indeed, might not be possible at all, depending on the type of convalescence the patient would undergo. Infection, though potential, had been staved off by chemotherapy and did not, at the moment, constitute a contraindication. The incision, therefore, was extended toward the midline, and, as was stated previously, the fourth costochondral junction was severed. The pericardium, which was about 2 mm. thick, was opened; it did not contain any fluid. If there had been any degree of hemopericardium, by this time either it had resorbed or the fluid had escaped through a temporarily patent laceration of the pericardium.

Palpation of the myocardium through the pericardial window revealed an elevated surface at the apex. The window was enlarged in order that this area might be visualized; a seared, friable, slightly necrotic myocardium presented itself about 2 cm. from the apex, in the left ventricle. Two chromic no. 1 surgical gut sutures were placed in the apex as tension and subsequent hemostatic sutures. A quick stab wound was made over the foreign body and a small hemostat inserted into the musculature to grasp it. The hemostat sank into the tissue for a distance of about 1.5 cm. and grasped the foreign body, which was immediately withdrawn.

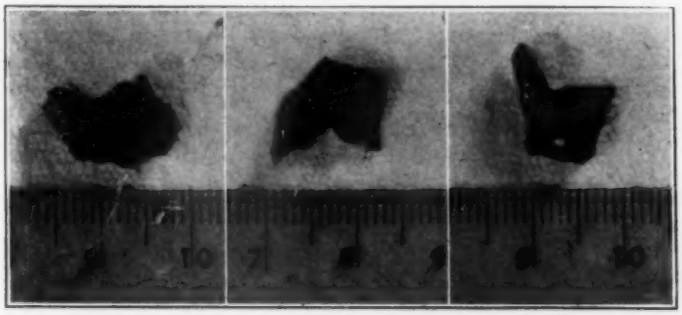

Fig. 3.—Three views of the shell fragment.

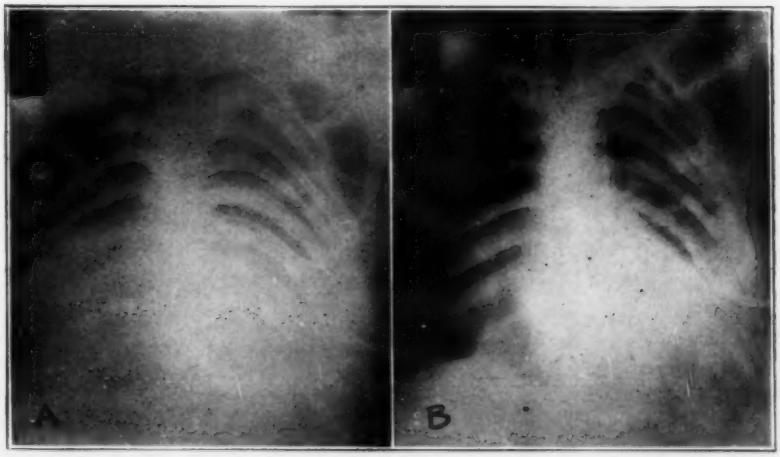

Fig. 4.—Roentgenograms of the patient in the anteroposterior position, taken with portable apparatus. *A*, three days after operation. The cardiac shadow is globular in shape and is displaced to the right. *B*, ten days after operation. The left cardiac border can now be identified.

It proved to be an irregular piece of shell fragment, with jagged edges, measuring 1.5 cm. by 1.2 cm. by 0.6 cm. (fig. 3). Concomitant with the stab wound, spurting bleeding ensued. After removal of the shell fragment, tying of the tension sutures helped control some of the bleeding, and, while traction was still maintained by them, two more sutures were inserted, producing complete hemostasis. The

pericardium was then irrigated with warm isotonic solution of sodium chloride, suctioned and closed, a window being left at the apical region for escape of whatever fluid might subsequently accumulate. Attention was then directed toward the closure of the thoracic wall, which was effected with waterseal, trap bottle drainage.

The patient was under anesthesia for one hour and forty-five minutes; the operation itself required one hour and ten minutes. The patient received 3 units of plasma during the operation and 600 cc. of whole blood immediately on return from the operating room, followed by 1,000 cc. of 5 per cent dextrose in isotonic solution of sodium chloride by hypodermoclysis and, later, a second 1,000 cc. intravenously. Oxygen, sulfadiazine and penicillin therapy were initiated at once, and an additional 500 cc. of whole blood was given the first postoperative day. Two hours after the operation the patient was taking fluids by mouth. His blood pressure was 130 systolic and 70 diastolic and gradually became stabilized at 116 systolic and 60 diastolic; in a few days it fell to 110 systolic and 60 diastolic, at which level it has since more or less remained.

About six hours after operation, a pericardial friction rub, audible over the right costocartilage area of the fourth rib developed. This lasted for about four hours and did not reappear. On the tenth day postoperatively, however, a blowing systolic murmur became evident over the third interspace, 2 cm. to the right of the midline; it occurred in no other region. The murmur diminished progressively and by the fifteenth postoperative day was entirely gone. With the exception of these two incidents, there have been no cardiac abnormalities discernible by physical examination.

Early postoperative complications of cardiac tamponade (due to nondraining pericarditis), atelectasis or pneumonia were watched for carefully but, fortunately, did not appear. Expansion of the left lung proceeded at a rapid rate (fig. 4). Of interest was the pronounced fulness of the right cervical vessels, indicating the shift and fixation of the mediastinum to that side. This was particularly noteworthy inasmuch as the right cervical region assumed an angulation of 90 degrees, in pronounced contrast to a smooth left cervical curvature at the juncture of the neck and shoulder region. This sign persisted until about the fifteenth postoperative day.

Serial electrocardiograms were taken from the day of operation (figs. 5 to 10). The cardiologist, Capt. Ellis W. Young, summarized them as follows:

"The essential changes noted in the records occurred in the ST segments and T waves. Tracings taken soon after operation showed elevated ST segments and small diphasic T waves. Later, as the ST segments approached the base level, coving was present and the T waves became inverted. In the final electrocardiogram the ST segments were isoelectric; the T waves were low and upright in leads I, II and III and inverted in lead IV F. These progressive variations are similar to those associated with pericarditis and are considered to represent the repair of a lesion of the myocardium following surgical treatment of the heart."

The sedimentation rate was 52 mm. per hour the first day postoperatively. By August 8, twelve days postoperatively, it was 11 mm. per hour. A final sedimentation rate, taken November 20, was 2 mm. per hour.

Except for a preoperative secondary anemia of 3,820,000 red blood cells and a 75 per cent hemoglobin content, which was overcome by transfusions, blood picture studies were of no special significance.

Six weeks following operation, the patient was permitted out of bed and graduated exercises were begun. Special exercises were

instituted early to assist in the reexpansion of the lung, to prevent deformity and to aid in the development of the thoracic cage. These exercises were carried out regularly four times daily, under the guidance of Lieut. Dora Dykins, the physical therapeutist, and were correlated with

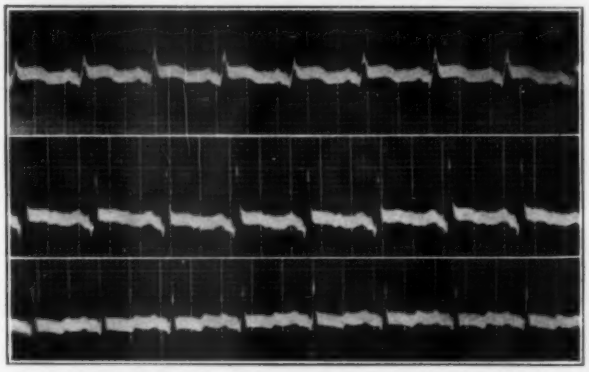

Fig. 5.—Electrocardiogram taken the first day postoperatively. The rate is 125, the P-R interval 0.12 seconds and the QRS interval 0.06 seconds. There are a slight elevation of the S-T segments and low voltage diphasic T waves.

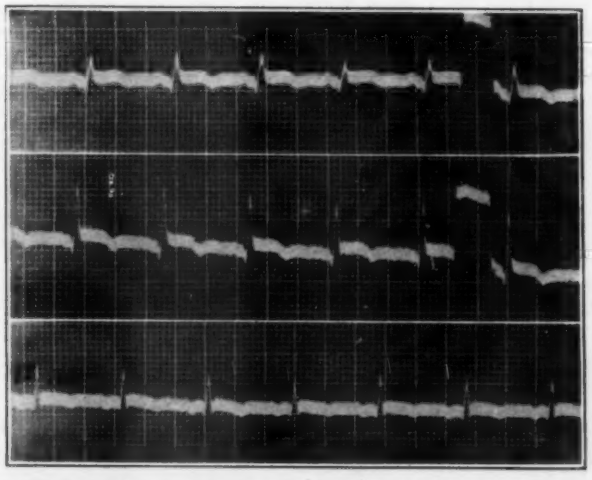

Fig. 6.—Electrocardiogram taken July 31, four days after the operation. The rate is 107, the P-R interval 0.10 seconds and the QRS interval 0.06 seconds. The T waves are low and are diphasic in lead I and inverted in leads II and III. The S-T segments are slightly elevated.

repeated flouroscopic examinations. The value of such exercises cannot be overemphasized.

At the time of this writing, four months after operation, the patient is in excellent condition, has no complaints and no deformity of the

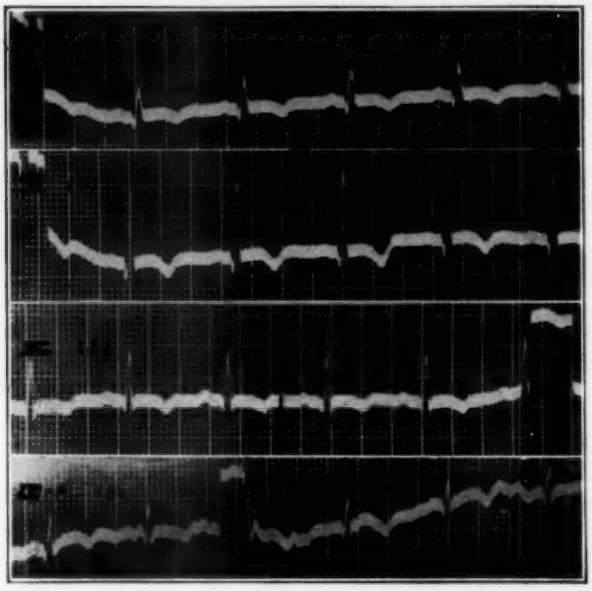

Fig. 7.—Electrocardiogram taken August 28, one month after the operation. The rate is 88, the P-R interval 0.14 seconds and the QRS interval 0.08 seconds. The T waves are inverted. The S-T segment is slightly elevated in leads I and II and isoelectric in lead III.

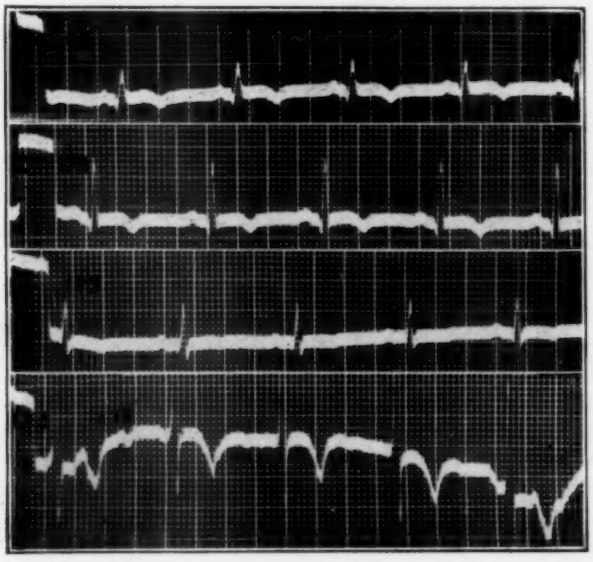

Fig. 8.—Electrocardiogram taken September 14. The rate is 80, the P-R interval 0.14 seconds and the QRS interval 0.08 seconds. The T waves are inverted in leads I, II and IV F and low and diphasic in lead III. The S-T segments are slightly elevated in lead I and isoelectric in leads II, III and IV F.

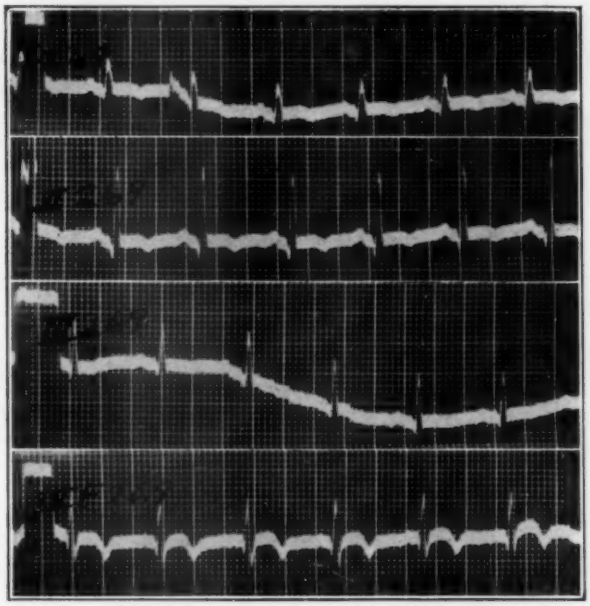

Fig. 9.—Electrocardiogram taken October 19. The rate is 105, the P-R interval 0.14 seconds and the QRS interval 0.07 seconds. The T waves have a lower voltage; they are inverted in leads I, II and IV F and diphasic in lead III. The S-T segments are practically isoelectric.

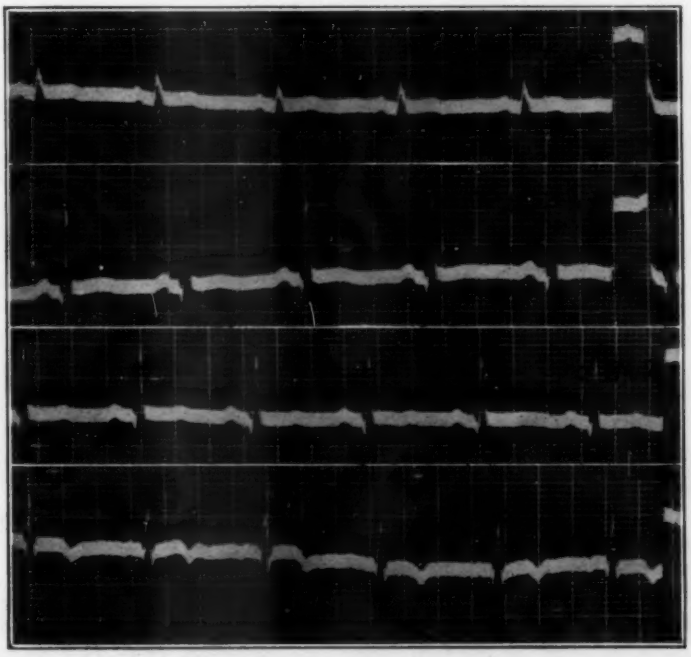

Fig. 10.—Electrocardiogram taken November 28, four months after operation. The rate is 84, the P-R interval 0.14 seconds and the QRS interval 0.08 seconds. The T waves are low and upright in leads I, II and III and inverted in lead IV F.

thoracic wall (fig. 12) and leads a completely independent sedentary existence, with progressive effort tolerance. His blood pressure is maintained at 110 systolic and 64 diastolic; the electrocardiograms indicate progressive improvement (fig. 10), and fluoroscopic follow-ups demonstrate continued improvement in the illumination of the lung

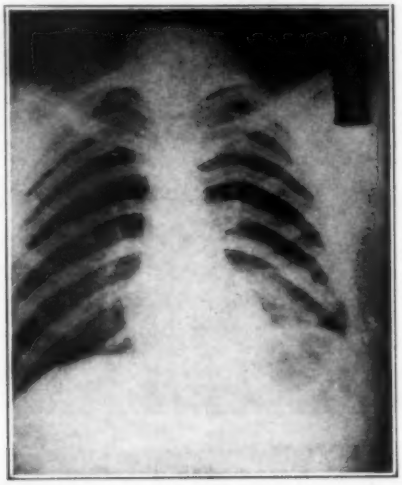

Fig. 11.—Roentgenogram taken November 28. The cardiac shadow is in the midline and is small. Lung fields show normal illumination except for some "streaklike" atelectatic markings of the lower left lobe. The left diaphragm is moderately elevated.

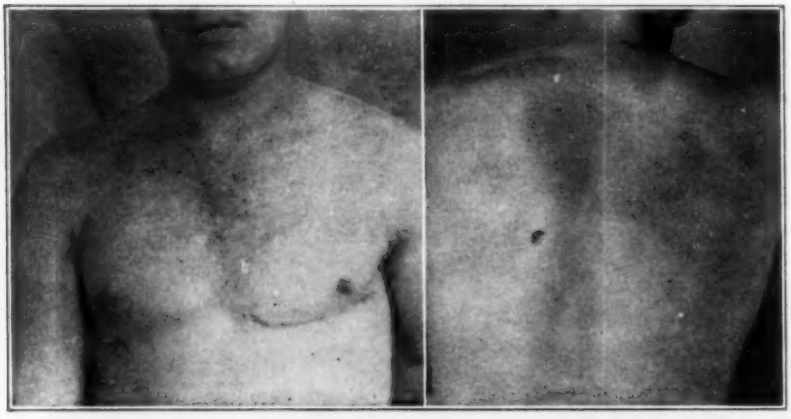

Fig. 12.—Pictures of patient taken three and one-half months after the operation, showing a well rehabilitated thorax. The wound of entrance is seen in the posterior view.

fields and in the motion of the costal cage and diaphragm. The heart is small, is normal in configuration and position (fig. 11) and shows good pulsation on fluoroscopic examination.

SUMMARY AND CONCLUSIONS

1. A case of successful removal of a shell fragment from the left ventricle of the heart is reported.

2. The value of overpenetrated roentgenograms and fluoroscopic examination in locating the foreign body, together with the definity of its edges as a diagnostic value, are noted.

3. Stress is laid on the importance of early and constant exercises for the rehabilitation of the chest.

4. Serial electrocardiograms showed progressive improvement of the myocardial lesion.

Captain Rakofsky, Captain Young and First Lieutenant Dykins, Medical Corps, Army of the United States, gave valuable assistance, and Technician Fifth Grade Charles H. Snyder, Medical Department, Army of the United States, aided with the photography for this article.

RECONSTRUCTIVE OTOPLASTY

Further Observations; Utilization of Tantalum Wire Mesh Support

CAPTAIN PAUL W. GREELEY (MC), U.S.N.R.

THE reproduction of an auricular cartilage still remains the greatest problem in the construction of a new ear. In an earlier paper,[1] personal experience with Gillies' method, utilizing a maternal ear cartilage as the architectural support around which to build a new ear, was described in detail. Subsequently, a total of fifteen ears were constructed by this procedure. At first, it appeared that this contribution, as described first by Gillies,[2] was a great step in the successful construction of this persistently difficult plastic operation. However, as time progressed, it became apparent that these transplanted auricular cartilages were not going to remain in their original conditions permanently. In each case, the individual cartilage underwent aseptic necrosis and was replaced by fibrous tissue. By and large, this transition developed in one and one-half to two years in small children and in six months to one year in patients of adolescent age. Consequently, with the fibrous tissue replacement, the inevitable contracture typical of all scar tissue ensued and the resulting ear shrank to a small irregular structure. Thus resulted an ear which was unsatisfactory to both the patient and the surgeon. When this fate was discussed with Gillies,[3] it was learned that his end results had been identical with those of my colleagues and me.

Just why the transplanted fresh isografts of auricular cartilage have undergone absorption is not clearly understood. Isografts of costal

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From the Division of Plastic Surgery, United States Naval Hospital, Oakland, Calif.

Read at the meeting of the American Association of Plastic Surgeons, Toronto, Canada, June 3, 1946.

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1. Greeley, P. W.: Reconstructive Otoplasty, Surgery **10**:457-462 (Sept.) 1941.

2. Gillies, H. D.: Reconstruction of the External Ear, Rev. de chir. structive, October 1937, pp. 169-179.

3. Gillies, H. D.: Personal communication to the author.

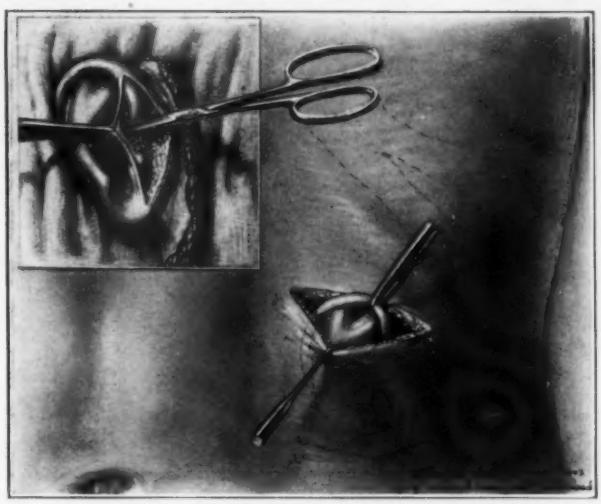

Fig. 1.—The insert in the upper left corner shows the cartilage being dissected from its overlying skin, the ear being entirely avulsed. Below is seen the pocketing of the cartilage in the anterior part of the abdominal wall. (Reproduced by the permission of the *United States Naval Medical Bulletin* **42**:1323-1325 [June] 1944.)

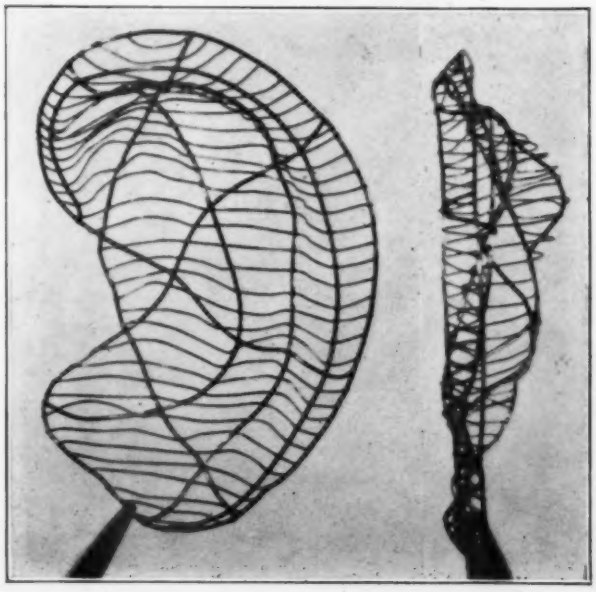

Fig. 2.—Two views of wire mesh model of ear.

Fig. 3.—Areas in temporomastoid skin covered with razor grafts to establish complete healing.

Thin skin flap raised. Tantalum mold inserted, flap sutured back.
1
2
3
4
5
Deep flap containing tantalum mold raised. Stent graft inserted and held in place by pressure dressing.

Fig. 4.—Operation showing burial of tantalum wire auricular cartilage mold beneath temporomastoid skin flap and coverage with stent graft.

cartilage on the whole behave satisfactorily. Likewise, transplantation of segments of the conchal portion of the cartilage to reconstruct a subtotal loss of the helix (homograft) is universally successful. Because of this behavior of homografts of auricular cartilage, it was suggested [4] in a previous paper that when one is confronted with a reasonably fresh

Fig. 5.—Result of operation shown in figure 4.

avulsion of all or part of an ear the cartilage should be preserved and buried subcutaneously until plastic reconstruction is started (fig. 1). While I have never had the opportunity to utilize this procedure, its theoretic application warrants special mention.

4. Greeley, P. W.: Reconstruction of the External Ear: Conservation of the Avulsed Portion, U. S. Nav. M. Bull. **42**:1323-1325 (June) 1944.

Other workers have made further contributions. Lamont,[5] in his paper, suggested the use of cartilage from a cadaver ear. In view of the foregoing experience, it would not seem likely that this method would be of success when a fresh isograft of auricular cartilage did not remain in its transplanted state permanently. Furthermore, neither Lamont nor other workers have described a case in which this method was actually carried out. Peer[6] described his method of using diced costal cartilage chips held together in a vitallium ear mold until fibrous tissue proliferation held the mass together. This excellent piece of work has promise.

USE OF TANTALUM

In the search for a better support, it was felt that the use of a tantalum frame might be practical. It is realized that tantalum is still a foreign body, but since it has been used successfully in a wide series of cases chiefly by orthopedic surgeons and neurosurgeons its apparent minimal reaction made its use seem worthy of trial in reconstructions of ears.

Through the cooperation of an engineer, Mr. Park B. Hyde, of the Fansteel Corporation, various models of ear cartilages were constructed. We eventually selected a fine wire mesh model, which not only was lighter but would permit development of circulation between the open spaces. These reproductions were made from a model of the normal ear. The wires were then shaped over a negative model by hand and the various pieces secured together by spot welding (fig. 2).

DESCRIPTION OF CASE

Our experience deals with only 1 actual case, but the result to date has been so satisfactory that it seems worthy of record for the benefit of persons who have not had an opportunity to see the patient.

An Aviation Ordnance Mate third class, aged 23, was struck and dragged by a station wagon, causing a complete avulsion of the left external ear and much of the adjacent surrounding normal skin. On admission to our service, three months after injury, three small granulating areas in the temporomastoid skin were covered with razor grafts to establish complete healing (fig. 3). In view of the amount of scarring in this area, the vascularity of the skin covering was extremely poor. Consequently, it was necessary to "delay" the primary skin flap six times before development of an adequate circulation was obtained in its base.

When the temporomastoid skin flap had sufficient vascularity, the tantalum wire mold of auricular cartilage was buried beneath it (fig. 4, part 2), after which the borders of the skin were sutured together. Primary healing ensued,

5. Lamont, E. S.: Reconstructive Plastic Surgery of Absent Ear with Necrocartilage: Original Method, Arch. Surg. **48**:53-72 (Jan.) 1944.

6. Peer, L.: Reconstruction of the External Ear with Diced Cartilage Grafts, Tr. Am. Soc. Plastic & Reconstruct. Surg., 1943, p. 11-16.

and the mold was permitted to remain untouched for seven months. This long interval was allowed so that one might be reasonably certain that no foreign body reaction might occur in this superficial location of relatively poor vascularity.

At the next stage, the curved incision in the skin flap was reopened, being dissected downward behind the transplanted tantalum frame, but carefully so as not to expose any segment of metal wire from the mass of fibrous tissue that had grown around the new support. The structure was then brought forward and the posterior surface of the new ear and the temporomastoid bed covered with a

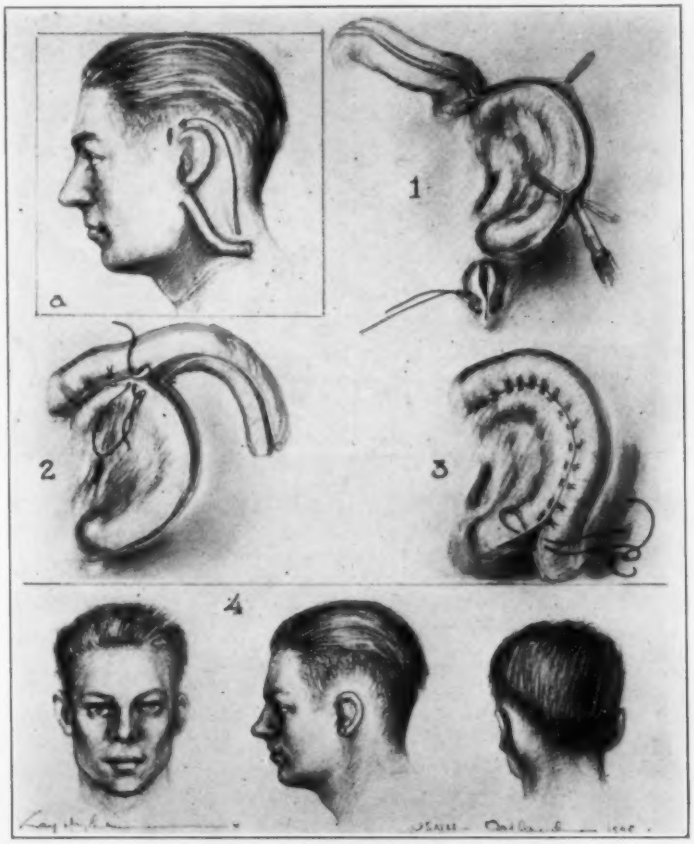

Fig. 6.—Operation showing addition of small tube pedicle flap.

stent graft such as has been described in other standard procedures (fig. 4, part 4). The final result is demonstrated in the artist's sketch (fig. 4, part 5) and by photographs in figure 5.

The new ear was followed closely, because it was felt that, although the procedure might have merit, complications might develop in this particular case because of the extensive fibrosis in both the anterior and the posterior coverings of the skin. Subsequently, after three months of complete healing and absence of reaction, two small areas of pressure

necrosis developed around the peripheral margin. After these areas were observed for six weeks, it seemed apparent that they developed because of scar contracture of the anterior and posterior coverings of the skin over the rigid periphery of the tantalum mold. However, no infection or other inflammatory reaction developed around the open areas.

Fig. 7.—Final result, twenty-five months after operation.

In order that the scarred periphery might be replaced with an elastic covering, this particular ear was supplemented with a small tube pedicle flap taken from the neck, as described by Pierce [7] (fig. 6). The present

7. Pierce, G. W.: Reconstruction of the External Ear, Surg., Gynec. & Obst. **50**:601-605 (March) 1930.

condition is shown in figure 7, twenty-five months following the original implantation of the tantalum frame.

COMMENT

It is realized that the newly constructed ear in the patient demonstrated is not architecturally ideal. This is due primarily to the extensive scarring in the skin bed from which the ear was made. Were the same procedure to be duplicated in skin of good vascularity, such as in a congenitally absent ear, a far superior result might be anticipated and the second stage utilized here be eliminated entirely.

However, the most important factor seems to be that an ear has been constructed around a tantalum wire model of a normal auricular cartilage. This tantalum structure has remained under the skin for a total of twenty-five months to date (Dec. 1, 1945), during which eighteen months represents the completion of a new ear. With the apparent successful behavior over a reasonably long period, the method should warrant trial by other plastic surgeons. This patient has been discharged from the Navy, but it is planned to follow his future progress in civil life.

SUMMARY

1. The subsequent unsuccessful results with Gillies' method of reconstructive otoplasty are discussed.

2. Reemphasis is made for preservation of the ear cartilage when one is dealing with a complete avulsion of the external ear.

3. A new method of reconstructive otoplasty is described, in which a tantalum wire model of an auricular cartilage has been successful and without reaction for over two years.

BLOCKING OF THE SPLANCHNIC NERVES AND THE FIRST LUMBAR SYMPATHETIC GANGLION

Technic, Accidents and Clinical Indications

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ANALYSIS of the evolution of surgery of the sympathetic system during recent years shows that some operations on this system in certain cases have enjoyed a period of enthusiasm followed by another which was less encouraging. In part, this phenomenon may be due to difficulty in determination of an accurate therapeutic indication in every case. The treatment of several diseases by operation on the sympathetic system is based fundamentally on the physiology and physiopathology of the sympathetic system. This being so wide knowledge of the physiology of the sympathetic innervation and of its relation to the disease or to the automatic disturbance is necessary before one decides on this operation. On the other hand, if it is possible before the operation to determine what result may be obtained by operation on the sympathetic system, this type of operation can attain a high degree of accuracy in its therapeutic indications and thus avoid operative failures. By blocking the sympathetic nerves, one can obtain a temporary interruption of their functions, which represents an important preoperative test in surgical measures on the sympathetic system.

In 1914 Kappis [1] introduced anesthesia of the splanchnic nerves, and in 1923 Läewen [2] demonstrated, by infiltrating procaine hydrochloride around the lower thoracic and upper lumbar white rami, that the sensory fibers carrying the painful sensations of the upper abdominal viscera run into the splanchnic nerves, and he determined also the levels at which these fibers enter the cord. Von Gaza [3] (1924) carried out investigations on the use of procaine hydrochloride to determine the pathways of obscure pain. In 1925 Mandl,[4] by para-

1. Kappis, M.: Erfahrungen mit Lokalanästhesia bei Bauchoperationen, Verhandl. d. deutsch. Gesellsch. f. Chir. **43**:87, 1914.

2. Läewen, A.: Weitere Ehrfahrungen über paravertebrale Schmerzaufhebung zur Differentialdiagnose von Erkrankungen der Gallenblase, des Magens, der Niere, und des Wurmfortsatzes, Zentralbl. f. Chir. **50**:461, 1923.

3. von Gaza, W.: Die Resektion der Paravertebralen Nerven und die isolierte Durchsneidung des Ramus communicans, Arch. f. klin. Chir. **133**:479, 1924.

4. Mandl, F.: Die Wirking der paravertebralen Injection bei Angina pectoris, Arch. f. klin. Chir. **136**:495, 1925.

vertebral anesthetic block, proved that sensory fibers from the heart run to the cord through the upper five or six thoracic rami. After these investigations, important contributions regarding sympathetic innervation and pain were made by Swetlow [5] (1926), Leriche [6] (1927) and Mixter and White [7] (1929). In 1930 White [8] showed that blocking of the sympathetic nerves with procaine hydrochloride is a useful diagnostic procedure to evaluate the quantitative increase in surface temperature that can be produced by sympathectomy and to determine the effect of sympathectomy on obscure pain referred over the sympathetic system. Since then, investigations have been carried out by Woodbridge [9] (1930), Morton and Scott [10] (1931), De Takats [11] (1931), Flothow [12] (1931), Leriche and Fontaine [13] (1934), Ruth [14] (1934), Leriche [15] (1940), Outland and Hanlon [16] (1940), Sousa Pereira [17] (1941), and Rovenstine and Wertheim [18] (1941) on anesthetic block of the sympathetic pathways as a method of diagnosis, a preoperative test or a method of treatment.

After I recognized the importance of the preoperative study of the innervation of the sympathetic system, I began performing its physiologic interruption as a test to determine the influence of block-

5. Swetlow, G. I.: Paravertebral Alcohol Block in Cardiac Pain, Am. Heart J. **1**:393, 1926.

6. Leriche, R.: La chirurgie de la douleur et ses resultats, Presse méd. **35**:497 and 561, 1927.

7. Mixter, W. J., and White, J. C.: Alcohol Injection in Angina Pectoris, Ann. Surg. **89**:199, 1929.

8. White, J. C.: Diagnostic Novocain Block of the Sensory and Sympathetic Nerves: A Method of Estimating the Results Which Can Be Obtained by Their Permanent Interruption, Am. J. Surg. **9**:264, 1930.

9. Woodbridge, P. D.: Therapeutic Nerve Block with Procaine and Alcohol, Am. J. Surg. **9**:278, 1930.

10. Morton, J. J., and Scott, W. J. M.: Methods for Estimating the Degree of Sympathetic Vasoconstriction in Peripheral Vascular Diseases, New England J. Med. **204**:955, 1931.

11. de Takats, G.: The Differentiation of Organic and Spastic Vascular Occlusions, Ann. Surg. **94**:321, 1931.

12. Flothow, P. G.: Diagnosis and Therapeutic Injections of the Sympathetic Nerves, Am. J. Surg. **14**:591, 1931.

13. Leriche, R., and Fontaine, R.: Technic of and Indications for Procaine Hydrochloride Infiltration of Lumbar Sympathetic, Presse méd. **42**:1843, 1934.

14. Ruth, H. S.: Diagnostic, Prognostic and Therapeutic Nerve Blocks, J. A. M. A. **102**:419 (Feb. 10) 1934.

15. Leriche, R.: Chirurgie de la douleur, ed 2, Paris. Masson & Cie, 1940.

16. Outland, T., and Hanlon, C. R.: The Use of Procaine Hydrochloride as a Therapeutic Agent, J. A. M. A. **114**:1330 (April 6) 1940.

17. de Sousa Pereira, A.: A technica dos bloqueios anestesicos dos nervos esplancnicos, Med. contemp. **60**: 1941.

18. Rovenstine, E. A., and Wertheim, H. M.: Therapeutic Nerve Block, J. A. M. A. **117**:1599 (Nov. 8) 1941.

ing the regional sympathetic system in each patient before operation. These anesthetic blocks were performed singly or were repeated in order to obtain a fairly accurate result. Then, in order to obtain a definitive result, previously ascertained by the physiologic interruption of the sympathetic ganglions or trunks, sympathectomy was performed. After I began following this routine, I observed in my cases an improvement in the results. This effect was especially important in the field of the splanchnic nerves to determine in every case the indications for splanchnicectomy. But in addition to being a preoperative test in this operation, single or repeated anesthetic blocks of the splanchnic nerves may also be a useful method of diagnosis or treatment in certain cases of visceral pain, vasomotor disturbances or disturbances of the tone of the smooth intestinal muscle. The importance of the therapeutic problems related to the splanchnic nerves led me to study these nerves especially. During the last eight years, more than one thousand anesthetic blocks of the splanchnic nerves or of these nerves and the first lumbar sympathetic ganglion have been performed with the technic that will be described. The accidents that have occurred and the indications for operation and the results in the cases that have been studied will also be described.

ROUTES OF APPROACH TO ANESTHESIA OF THE SPLANCHNIC NERVES

Kappis [1] was the first to introduce anesthesia of the splanchnic nerves, having made his preliminary report in 1914 to the Congress of Surgery in Berlin. In 1918 he published a report of a series of 200 cases. To reach the nerves he followed a posterior route. This technic will be described later.[19] In the same year Wendling [20] suggested the approach to the splanchnic nerves by the anterior route, by introducing a long needle through the intact anterior abdominal wall a little below and to the left of the ensiform cartilage. This needle, to reach the retroperitoneal nerves and ganglions, must cross the left lobe of the liver and the lesser omentum. It was demonstrated to be an inaccurate and dangerous method and was quickly abandoned. Braun,[21] in 1919, intro-

19. Kappis, M.: (*a*) Die Anästhesierung des Nervus splanchnicus, Zentralbl. f. Chir. **45**:709, 1918; (*b*) Sensibilität und lokale Anästhesie in chirurgischen Gebiet der Bauchhöhle mit besonderer Berücksichtigung der Splanchnicus-Anasthesie Beitr. z. klin. Chir. **155**:161, 1919; (*c*) Zur Technik der Splanchnicusanästhesie, Zentralbl. f. Chir. **47**:98, 1920; (*d*) Ueber Splanchnicus anaesthesie, Verhandl. d. deutsch. Gesellsch. f. Chir. **44**:275, 1920; (*e*) Die Technik der Einspritzungen den Nervus splanchnicus, Zentralbl. f. inn. Med. **46**:1097, 1925.

20. Wendling, H.: Ausschaltung der Nervi splanchnici durch Leitungsanästhesie bei Magenoperationen und andern Eingriffen in der oberen Bauchhöhle, Beitr. z. klin. Chir. **110**:517, 1918.

21. Braun, H.: Ein Hilfsinstrument zur Ausführung der Splanchnicusanästhesie, Zentralbl. f. Chir. **48**:1544, 1921.

duced direct anesthesia of the splanchnic nerves and the solar plexus after a previous laparotomy.

In cases in which it is necessary to anesthetize the abdominal viscera, a previous laparotomy having been performed, anesthesia of the splanchnic nerves can be obtained by following Braun's technic. After a supraumbilical median or paramedian laparotomy has been performed, the left lobe of the liver is retracted upward and the stomach pulled carefully downward and to the left. Thus the lesser omentum is exposed, and with the left forefinger through the omentum one tries to palpate the aorta, which is recognized by its pulsation. In its progress toward the spine, the left forefinger pushes the aorta to the left and the vena cava to the right. A fine needle 12 cm. long is introduced through the lesser omentum so that it passes over the pancreas and between the two blood vessels which are held apart by the left forefinger. The needle goes through the retroperitoneal tissue up to the crura of the diaphragm. After first aspirating to make sure that the needle is not in a blood vessel, one proceeds to inject into the loose prevertebral periaortic tissue 100 cc. of 0.5 per cent solution of procaine hydrochloride. To obtain an easier injection, various mechanical devices have been tried. Kirchmayer,[22] Burke [23] and Denman [24] have described conductors and protecting tubes to direct the needle along the finger.

Injection by the posterior route, described by Kappis,[19] was the first to be put into practice. Its superiority is shown by the fact that it permits one to anesthetize the splanchnic nerves by the lumbar route merely by the introduction of a conveniently directed needle. Here is Kappis' description: The patient lies prone, with a pillow under the costal arch to bend the vertebral column. Seven centimeters from the midline and just below the twelfth rib, a needle 12 cm. long is directed so as to form an angle of 30 degrees with the sagittal plane. When the needle reaches the depth of the vertebral body, one slides it tangentially to the border of the vertebra until its tip has lost contact with the bone, which corresponds to an advance of about 1 cm. Then, after one has aspirated with the syringe to see whether the needle has penetrated a blood vessel, 20 to 40 cc. of 1 per cent solution of procaine hydrochloride is injected. To obtain complete anesthesia, one proceeds similarly in relation to the splanchnic nerves on the opposite side. The bilateral anesthetic blocks are repeated, with a similar technic, at the height of the second lumbar vertebra.

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22. Kirchmayer, L.: Eine gedeckte Nadel zur Splanchnicusanästhesie nach H. Braun, Zentralbl. f. Chir. **50**:1480, 1923.

23. Burke, J.: A New Instrument in Splanchnic Anesthesia, J. A. M. A. **82**:790 (March 8) 1924.

24. Denman, P. R.: A New Instrument in Splanchnic Anesthesia, J. A. M. A. **84**:1179 (April 18) 1925.

In 1920 Kappis [19c, d] began to employ only one injection on either side of the vertebral column. The needle is inserted below the twelfth rib, and 20 cc. of the procaine hydrochloride solution is injected; after this, the needle is directed upward and downward, parallel to the vertebral bodies, and 20 cc. of the solution is injected in either direction. Later Kappis advised only one injection on either side but without the upward and downward slant of the needle.

Labat [25] also described a technic of anesthesia of the splanchnic nerves by a posterior route, similar to that of Kappis. He modified the inclination of the needle from an angle of 30 degrees to one of 45 degrees. Roussiel [26] followed also a posterior approach and named this method paravertebral anesthesia.

Two technics of anesthesia of the splanchnic nerves must be kept in mind. In cases in which previous laparotomy has been performed, Braun's technic, by the anterior route, is indicated. The technic of Kappis, by the posterior route, permits injection of anesthetic solution around the splanchnic nerves through a needle introduced just below the last rib and directed toward the anterolateral region of the first lumbar vertebra. This method does not require a previous laparotomy.

De Takats,[27] in 1927, made a complete study of both methods of anesthetizing the splanchnic nerves, anterior and posterior. From the technical point of view, the problem consists in infiltration of the loose connective tissue which lies in front of the first lumbar vertebra with the anesthetic solution, and this can be done by either the anterior or the posterior route. He demonstrated that experimental and clinical facts showed that bilateral anesthetic block of the splanchnic nerves leads to anesthesia of the upper abdominal organs. Comparing the results of anesthesia induced by injections by a posterior approach, obtained in 2,335 cases, with those of anesthesia induced by injections by an anterior route, obtained in 1,222 cases, de Takats concluded that the injection by the anterior route yielded better results and carried less risk than that by the posterior one. It is true that the anterior route permits injection of the anesthetic solution close to the splanchnic nerves and the solar plexus directly and under control of the finger. As it necessitates a previous laparotomy, this technic is indicated in only a

25. Labat, G.: L'anesthésie splanchnique dans les interventions chirurgicales et dans les affections douloureuses de la cavité abdominale, Gaz. d. hôp. **93**:662, 1920; Regional Anesthesia with Special Reference to Splanchnic Analgesia, Brit. J. Surg. **8**:278, 1921; The Induction of Splanchnic Analgesia, Ann. Surg. **80**:161, 1924; Regional Anesthesia, Philadelphia, W. B. Saunders Company, 1922, p. 350.

26. Roussiel, M.: Anesthésie des nerfs splanchniques et des plexus mésentériques supérieurs et inférieurs en chirurgie abdominal, Presse méd. **31**:4, 1923.

27. de Takats, G.: Splanchnic Anaesthesia: A Critical Review of the Theory and Practice of This Method, Surg., Gynec. & Obst. **44**:501, 1927.

limited number of cases. In all cases in which it is necessary to perform anesthetic block of the splanchnic nerves without previous laparotomy, only the posterior route is suitable. My colleagues and I have seen that repeated punctures in the same place and at short intervals in order to block the splanchnic nerves by the posterior route quickly become painful and unbearable by the patient. Observation of this fact led me to study a technic that would be better tolerated by patients.

ANATOMIC FACTS ON WHICH IS BASED THE TECHNIC OF BLOCK OF THE SPLANCHING NERVES BY THE POSTERIOR ROUTE

Dissection and study of the splanchnic nerves, the solar plexus and the thoracolumbar sympathetic chain in 100 human cadavers led me to the following conclusions regarding the morphology and topography of these nerves.[28]

The splanchnic nerves establish the connection between the thoracic sympathetic chain and the solar plexus (fig. 1). Normally two splanchnic nerves effect this connection (53 per cent of cases). Less often (43 per cent) there are three splanchnic nerves. In rare cases (4 per cent) there are four splanchnic nerves. These are the greater splanchnic, the lesser splanchnic, the lowest splanchnic and the accessory splanchnic. The greater splanchnic nerve originates in the segment of the thoracic sympathetic chain lying between the fourth and eleventh thoracic ganglion. It is usually formed by three branches or, less often, by four or five. It may also be constituted by as few as one or as many as seven branches. Most frequently the branches of the greater splanchnic nerve come from the seventh, eighth and ninth thoracic sympathetic ganglions. These branches run downward and obliquely inward on the anterolateral aspect of the spine and come together at various levels to form a cord, which in its downward course passes over the anterolateral aspect of the body of the last thoracic vertebra. It perforates the crura of the diaphragm and divides into its terminal branches, ending in the semilunar ganglion. At the origin of the trunk of the greater splanchnic was found in about 70 per cent of cases a small ganglion (splanchnicum ganglion), normally opposite the level of the eleventh or twelfth thoracic vertebrae.

The greater splanchnic nerve pierces the diaphragm usually (63 per cent) through the space which separates the internal from the external crus of the vertebral insertion of the diaphragm. In other cases (13 per cent) this nerve perforates directly the single crus. The internal crus is perforated in 9 per cent of cases and the external crus in 3 per cent, or the nerve passes through the space between the internal and inter-

28. de Sousa Pereira, A.: Nervi splanchnici, Thesis, Oporto, Portugal, 1929.

medial crura as verified in 6 per cent of cases. Only rarely (3 per cent) does the greater splanchnic nerve penetrate the abdomen by the aortic hiatus. Whatever the route followed by the greater splanchnic nerve across the diaphragm, after it enters the abdominal cavity it is directed obliquely downward and inward or, at other times, transversely inward and divides into various terminal branches, which spread out like a fan. A great number of these fibers go to the semilunar ganglion, where they terminate; others are directed to the aorticorenal

Fig. 1.—Abdominal and pelvic sympathetic innervation: *1* and *6*, adrenal glands; *2*, greater splanchnic nerve; *3*, adrenal sympathetic fibers coming from the solar plexus; *4*, renal plexus; *5*, superior hypogastric plexus; *7*, solar plexus; *8*, aorticorenal ganglion; *9*, aortic plexus; *10*, lumbar sympathetic chain; *11*, inferior hypogastric plexus.

ganglion or to the cord which connects this ganglion with the semilunar ganglion.

The lesser splanchnic nerve is usually formed by two branches—sometimes by one or by three—from the thoracic sympathetic chain, between the tenth and twelfth thoracic ganglions. Soon after their origin these branches converge into one trunk, which descends down-

ward, inward and forward along the lateral aspect of the spine, between the greater splanchnic nerve and the sympathetic chain. When it reaches the diaphragm, the lesser splanchnic nerve usually insinuates itself into the interstice which separates the internal and external crura (68 per cent), under the greater splanchnic nerve and in front of the sympathetic chain. Less often it perforates the single crus (6 per cent), the internal crus (3 per cent) or the external crus (2 per cent) or passes between the internal and intermedial crura (7 per cent). In some cases it enters the abdominal cavity through the aortic hiatus (9 per cent). The lesser splanchnic nerve, after entering the abdomen, normally emerges from the diaphragmatic crura 1 or 2 cm. below the greater splanchnic nerve. From its point of entry into the abdominal cavity to its ending in the aorticorenal ganglion or the semilunar ganglion, to pass to the renal plexus the lesser splanchnic nerve generally follows an obliquely downward course, sometimes a transverse one and rarely a recurrent course.

The lowest splanchnic nerve, observed in 43 per cent of cases, is usually made up of a single branch or, rarely, of two branches which originate from the ganglions or from the intermediary cord of the sympathetic system, between the tenth and twelfth thoracic ganglions. This nerve, which is usually an extremely fine cord situated behind and below the lesser splanchnic nerve and in front of the sympathetic chain, follows a nearly rectilineal course from its origin to the diaphragm between these two nerves or between the lesser splanchnic nerve and the accessory splanchnic nerve when it exists. It normally enters the abdomen by the space between the internal and external crura of the diaphragm, below the greater and the lesser splanchnic nerves and above the lumbar sympathetic chain. Only rarely does it perforate the single crus, the internal crus or the external crus or pass through the aortic hiatus. In the abdominal cavity the lowest splanchnic nerve extends toward the origin of the renal artery and almost always terminates in the aorticorenal ganglion.

The accessory splanchnic nerve, which I have found 4 times in 100 cases, is a fine cord, independent of the lowest splanchnic nerve but with a similar course. Originating from the eleventh or the twelfth thoracic ganglion, it descends alongside the lowest splanchnic nerve, with similar anatomic relations, and ends in the aorticorenal ganglion.

The splanchnic nerves normally terminate at the external border of the solar plexus, which is formed by a series of different-sized ganglions anastomosed among themselves by numerous cords (fig. 1). The solar plexus, a network of ganglions in which some authors describe three groups of symmetric ganglionic masses (semilunar, superior mesenteric and aorticorenal ganglions), presents a variable disposition, not fitting into any rigid pattern. In my dissections I found that the

nerve ganglions and the cords that connect them usually form a flat, more or less tightly meshed network, extending transversely in front of the aorta and surrounding the celiac artery and the root of the superior mesenteric artery. In the transverse plane the solar plexus occupies the space between the two adrenal glands, passes beyond the lateral borders of the aorta on both sides and rests in front of the crura of the diaphragm and the commencement of the abdominal aorta (fig. 1). The greater splanchnic nerve, after crossing the crura of the diaphragm, ends in the upper pole of the lateral border of the solar plexus. In many cases, at this level there exists a ganglion mass which is more voluminous than the neighboring ganglions and which is generally called the semilunar ganglion. The lesser splanchnic nerve, the lowest splanchnic nerve and the accessory splanchnic nerve terminate at the external border of the solar plexus, at points which vary from case to case but which lie in a region situated between the semilunar and aorticorenal ganglions.

The solar plexus occupies an area 2 or 3 cm. high by 3 or 4 cm. wide between the origin of the celic artery and that of the renal arteries. From it emerge secondary plexuses, which accompany the celiac artery (celiac plexus), the superior mesenteric artery (superior mesenteric plexus), the phrenic arteries, (phrenic plexus), the suprarenal arteries (suprarenal plexus) and the renal arteries (renal plexus). Normally situated in front of the first lumbar vertebra, the upper border of the solar plexus in some cases extends on to the inferior portion of the last thoracic vertebra, and in some cases the lower portion of the plexus corresponds to the second lumbar vertebra. In relation to the soft parts, the upper limit corresponds to the aortic hiatus of the diaphragm and the lower limit corresponds to the origin of the renal arteries. Situated in front of the aorta and the crura of the diaphragm, above the pancreas and within the internal borders of the suprarenal glands, the solar plexus covered in front by the peritoneum, lies in a connective tissue, rich in fat, in which there are lymphatic ganglions and numerous lymph and blood vessels. Caudalward the solar plexus is continuous with the aortic plexus, from which arises part of the spermatic, the inferior mesenteric and the hypogastric plexuses (fig. 1). It is known at the present time that the splanchnic nerves carry the sensory fibers for a large group of abdominal organs. Anesthetic block of the splanchnic nerves results in anesthesia of the organs innervated by these nerves.

MORPHOLOGY AND TOPOGRAPHY OF THE TWELFTH RIB: ITS RELATION TO THE POSTERIOR ROUTE OF ANESTHETIC BLOCK

According to the technic of anesthesia of the splanchnic nerves induced by the posterior route, described by Kappis, the needle must be

inserted 7 cm. from the midline, just below the inferior border of the twelfth rib, and must be directed in such a way that it permits injection of the anesthetic solution close to the splanchnic nerves and the solar plexus. If the morphology of the last rib were always the same and if the angle formed by this rib with the medium plane were invariable, the reference point established by Kappis should be accurate. Such, however, is not the case. On the contrary, it is known that the solar plexus and the terminal portion of the splanchnic

Fig. 2.—Topography of the splanchnic nerves and solar plexus in relation to the skeleton.

nerves generally correspond to the first lumbar vertebra (fig. 2), and the renal arteries are found normally at the level of the second lumbar vertebra. The anesthetic solution should be injected above these vessels, in particular at the level of the first lumbar vertebra.

The point of the lower border of the twelfth rib, which is 7 cm. from the midline, differs with the angle of inclination of the last rib. This angle varies within wide limits (fig. 3) not only from case to case but from the right side to the left side in the same person (see the

table). In twenty roentgenograms of the thoracolumbar portion of the spine and the twelfth rib, I have studied the angle formed by the last rib with the lumbar portion of the spine and also the total length of this rib. The results are given in the table. Thus I have demonstrated that the angle formed by the inferior border of the twelfth rib and the lumbar portion of the spine varies from 30 to 70 degrees. The angles most frequently found were between 40 and 55

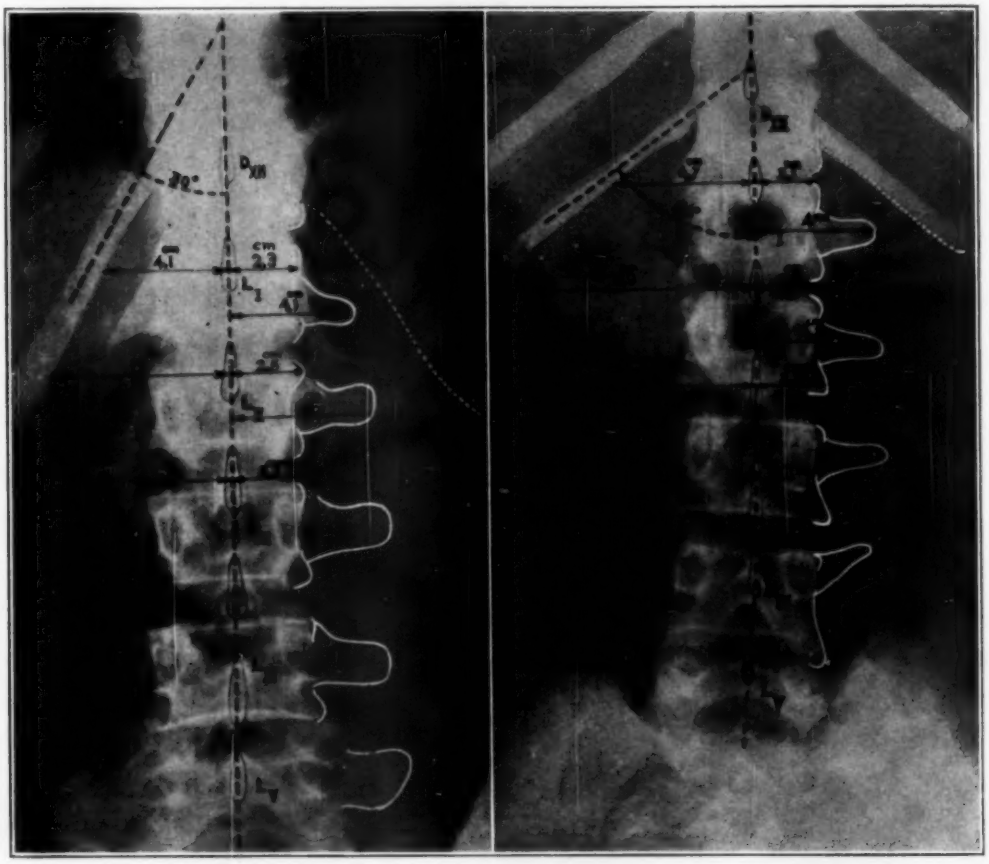

Fig. 3.—Roentgenograms and diagrams which show the difference in the angle of incidence of the lower border of the twelfth rib in relation to the axis of the spine. Note also the width of the vertebral bodies and the length of the transverse processes in relation to the midline.

degrees. These observations showed that in the majority of cases a horizontal line traced through a point situated 7 cm. from the midline in the lower border of the last rib corresponds to the second lumbar vertebra. Only in rare cases was it situated in front of the first lumbar vertebra. It also must be remembered that the length

of the twelfth rib is variable (fig. 4). In the 20 cases studied, the twelfth rib had lengths ranging from 3 to 15 cm. On the right side ribs 10 cm. long were found, and on the left side the length varied from 6 to 13 cm. This being so, not only must the angle of the twelfth rib be previously determined by palpation or roentgenologic examination but also accurate information on the length of the last rib must be obtained.

AREA WHERE BLOCK OF THE SPLANCHNIC NERVES IS PRACTICED

Several technics have been described (Kappis [19] and Labat [25]) for block of the splanchnic nerves with anesthetic infiltration by the pos-

Measurements of Twelfth Rib as Taken from Twenty Roentgenograms

Number of Cases	Angle Formed by the Right 12th Rib, Degrees	Angle Formed by the Left 12th Rib, Degrees	Length of the Right 12th Rib. Cm.	Length of the Left 12th Rib. Cm.
1	35	33	15	13
2	60	40	5	10
3	55	40	12	11
4	50	55	4	6
5	35	45	9	9.5
6	55	50	10	9
7	55	35	10	9.5
8	55	60	10	10.5
9	40	45	5.5	3
10	40	45	12	10
11	60	55	4.5	5
12	40	45	12.5	11.5
13	45	55	8	7
14	50	45	10	12
15	70	65	10	9
16	45	45	11.5	7
17	55	35	10	12
18	50	40	11	13
19	54	48	8	6
20	30	42	14	11.5

terior route. When these technics are followed, the needle is introduced into a preestablished point, located 7 cm. from the midline, just below the lower border of the twelfth rib. However, study of the morphology and topography of the splanchnic nerves in relation to the last rib showed that variations exist from case to case. Also in patients on whom I have employed these technics I have observed that punctures in the same place and repeated at short intervals are painful and badly tolerated. These two facts led me to try to develop a technic that would permit one to perform repeated, even daily, anesthetic blocks of the splanchnic nerves and sympathetic chain, without the procedures becoming unbearable. I tried to determine not a point, as Kappis did, but an area in which it was possible to introduce a needle, always at

different points, and to direct it so that the terminal portion of the splanchnic nerves was infiltrated by the anesthetic solution.

From my investigations I have concluded that the best area is represented by a triangle (fig. 5) which is bounded internally by the body of the first lumbar vertebra and by the upper half of the body of the second lumbar vertebra, above by the lower border of the twelfth rib and below by a transverse plane passing midway between the spinous processes of the first and second lumbar vertebra. The terminal portion of the splanchnic nerves and the external border of the solar plexus correspond in depth to the internal side of this triangle (fig. 6).

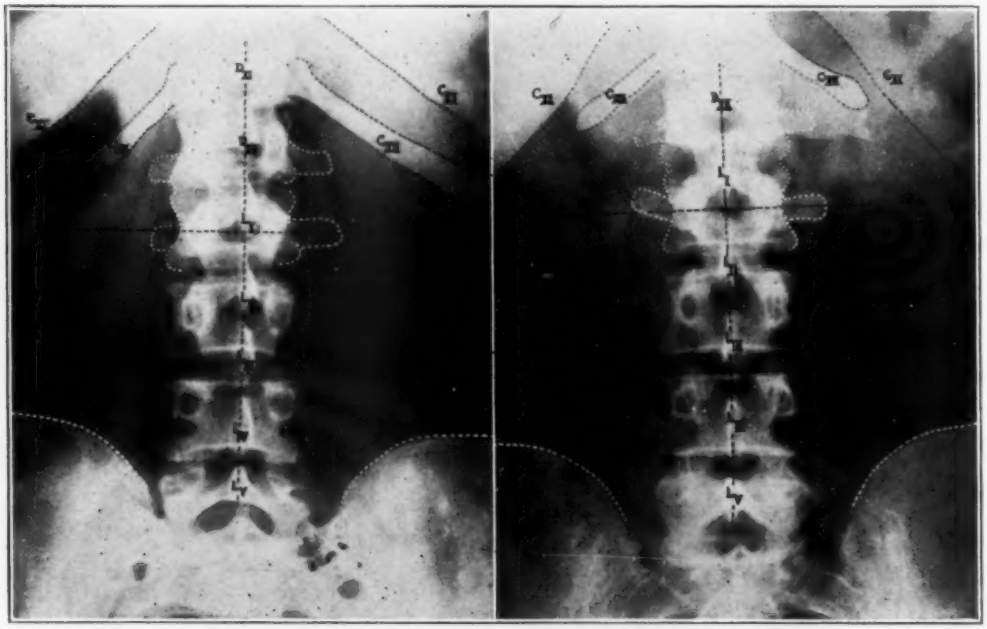

Fig. 4.—*A*, morphology of the twelfth rib and triangle formed by the transverse plane passing at the level of the transverse processes of the second lumbar vertebra. *B*, morphology of the twelfth rib and triangle formed by the transverse plane passing at the level of the transverse processes of the second lumbar vertebra.

TECHNIC OF ANESTHETIC BLOCK OF THE SPLANCHNIC NERVES AND THE FIRST LUMBAR GANGLION

After previous determination of the arterial blood pressure, the patient is placed in a sitting position, with the dorsolumbar region exposed. He flexes his trunk and rests the hands on his thighs or on the edge of the bed (fig. 7). If his condition does not permit this position, he is placed in the lateral decubitus position, back arched and knees flexed. By inspection, palpation or roentgenologic examination, an exact idea of the topography of the last rib and of the spinous proc-

esses of the first and second lumbar vertebrae is acquired. A transverse plane midway between these two processes and passing laterally until the lower border of the twelfth rib is reached is drawn. This plane normally corresponds to the transverse process of the second lumbar vertebra. Within the triangle bounded inferiorly by this plane, above by the lower border of the twelfth rib and internally by the lateral aspect of the body of the first and second lumbar vertebrae, an

Fig. 5.—Triangle in which the punctures for splanchnic nerve block should be done. This triangle is bounded internally by the external border of the first and second lumbar vertebrae, above by the lower border of the last rib and below by a transverse plane passing halfway between the spinous processes of the first and second lumbar vertebrae. In depth this plane corresponds to the transverse processes of the second lumbar vertebra.

area remains in which it is possible to insert a needle that will carry the anesthetic close to the splanchnic nerves (fig. 7).

The inner limits for the insertion of the needle is 2.5 cm. from the midline. If the needle is introduced at this distance it must be directed parallel to the sagittal plane and pass above or below the transverse

process of the first lumbar vertebra (figs. 8 and 9). Once the needle has passed at a tangent to the body of the vertebra, it is advanced 1 to 2 cm. farther. At this point it is felt that the needle, after piercing the muscular aponeurotic plane, enters in a free space. Its tip is then in the loose connective tissue which surrounds the terminal portion of the splanchnic nerves and the external border of the solar plexus. Aspiration is made with the syringe to make sure that the needle has not penetrated a blood vessel. Then 30 to 35 cc. of a 1 per cent anesthetic

Fig. 6.—Deep topography of the triangle (fig. 5) in the area of which must be inserted the needle to produce the anesthetic block of the splanchnic nerves and first lumbar sympathetic ganglion: *1*, solar plexus; *2*, greater splanchnic nerve; *3*, lesser splanchnic nerve; *4*, lumbar sympathetic chain; *5*, sacralis sympathetic chain; *6*, adrenal gland; *7*, semilunar ganglion; *8*, renal plexus; *9*, superior hypogastric plexus.

solution of procaine hydrochloride is injected slowly, so as to avoid the sudden dissociation of the tissues that may cause some rupture of small vessels and give rise to a hematoma. Several times during the injection, aspiration is done to make sure that the needle is not in a vessel. The other points at which the needle may be introduced are

situated within the area of the aforementioned triangle (fig. 7), between its internal border and the lower border of the last rib. When this rib is approached, it is necessary to increase progressively the angle of inclination of the needle in relation to the sagittal plane so that the needle passes deeply at a tangent to the body of the vertebra (figs. 8 and 9). The needle is made to progress 1 to 2 cm., and in the same way, once the musculoaponeurotic plane is passed, the point of the needle gives the sensation of less resistance, which shows that it has reached the loose connective tissue in which the splanchnic nerves are located.

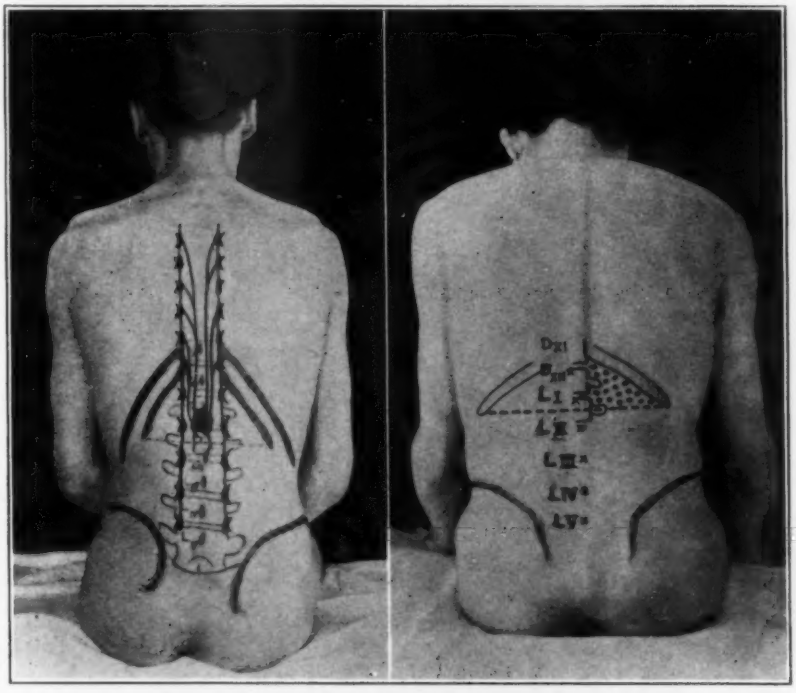

Fig. 7.—Demarcation of the triangle within which the needle must be inserted to perform the anesthetic block of the splanchnic nerves. Topography of the splanchnic nerves in relation to the skeleton and sitting position of the patient for the anesthetic block.

To block especially the first lumbar sympathetic ganglion and the splanchnic nerves by the same approach (fig. 10), the needle is inserted in the area of the triangle above the transverse process of the first lumbar vertebra. From 2.5 cm. of the midline to the lower border of the twelfth rib the needle may be introduced in any place within the area of the triangle (fig. 7). The needle is inserted parallel to the sagittal plane or obliquely, according to the place of puncture. It must be directed in order to pass deeply tangential to the body of the

first lumbar vertebra. After the needle is so directed, it is introduced about 1 cm. more and one begins injecting 3 to 5 cc. of the anesthetic solution into the musculoaponeurotic tissue at the same time that the needle is slowly introduced until one feels that its point is free and that the anesthetic solution diffuses quickly and easily into the prevertebral loose connective tissue. When the point of the needle attains this plane in front of the body of the vertebra, 5 cc. more of the anesthetic solution is injected in this plane. This anesthetic solution blocks the

Fig. 8.—The two sections shown in figure 9 were done, one, *A*, 0.5 cm. above the transverse processes of the first lumbar space and the other, *B*, at the level of the transverse processes of the second lumbar space.

sympathetic chain at the level of the first lumbar ganglion that is placed against the body of the first lumbar vertebra. The needle is then introduced 1 to 2 cm. farther into the loose connective tissue that surrounds the terminal portion of the splanchnic nerves. Into this tissue 25 cc. of 1 per cent solution of procaine hydrochloride is injected. If the needle is inserted below the transverse process of the first lumbar vertebra in the area of the same triangle (fig. 7), it must be directed

upward in order to reach the first lumbar ganglion and the splanchnic nerves in front of the first lumbar vertebra.

After one gains experience with the technics described, the needle may be quickly introduced near the body of the vertebra, without touching the periosteum. It is important to avoid punctures of the bone,

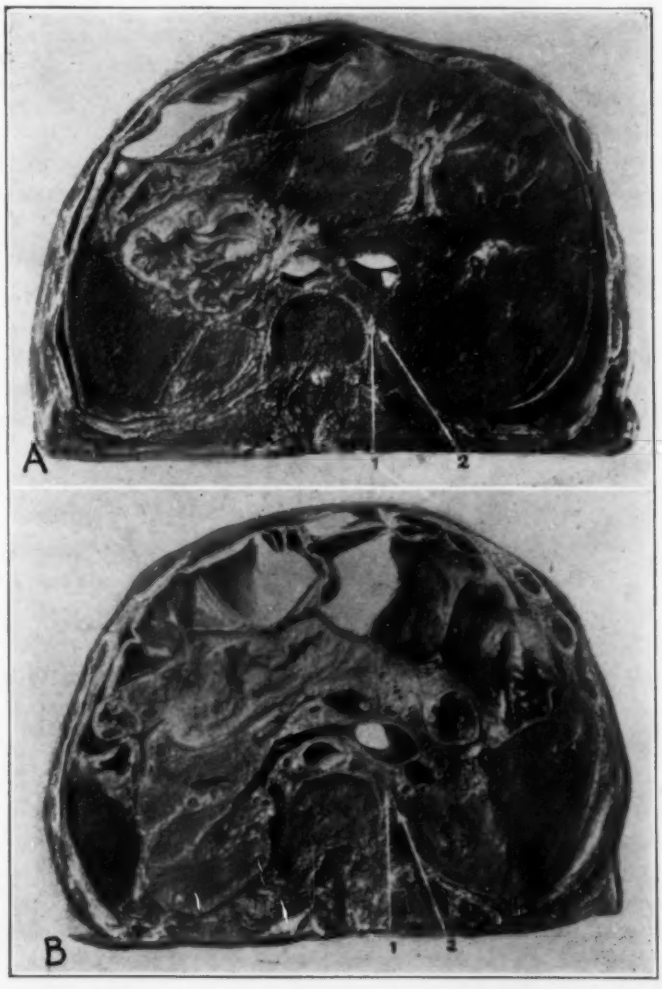

Fig. 9.—Directions which should be given to the needle (*1* and *2*) according to the point of puncture so as to reach the abdominal segment of the splanchnic nerves. *A*, transverse section passing 0.5 cm. above the superior border of the transverse processes of the first lumbar space. *B*, section at the level of the transverse processes of the first lumbar space and catching the renal pedicle.

because they are painful. When repeated anesthetic blocks of the splanchnic nerves are necessary, it is possible to change the point of insertion of the needle in the area of the triangle (fig. 5). Repeated

punctures, always at different points, are easily tolerated by the patient. Using the technic described, I have performed repeated anesthetic blocks of the splanchnic nerves alone or of these nerves and the first lumbar ganglion. The results were controlled by study of the arterial blood pressure and the degree of glycemia. The increase in temperature and the changes in cutaneous resistance at the level of

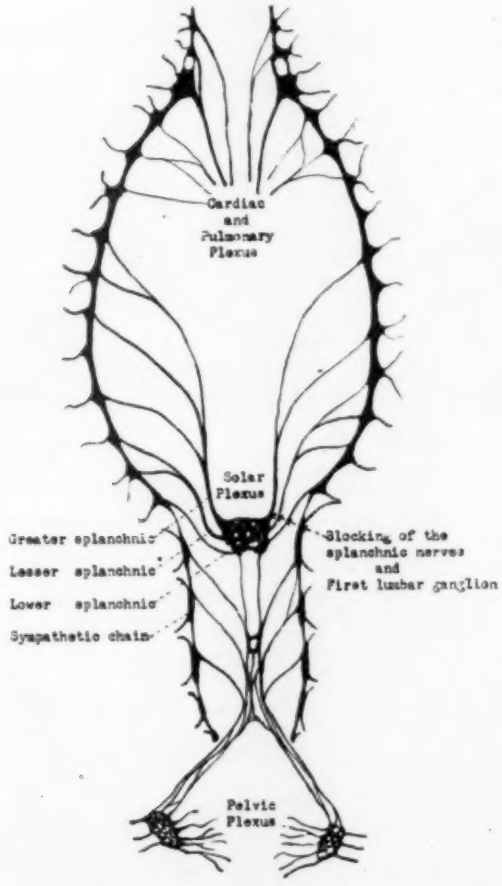

Fig. 10.—Block of the first lumbar sympathetic ganglion and the splanchnic nerves by the same approach.

the lower extremity showed the result of the anesthetic block of the first lumbar ganglion of the sympathetic chain. The results that I have obtained in a large series of cases using that technic justify the description of the technic. For my co-workers and myself, this technic was normally followed by successful results and was well tolerated by the patients.

ACCIDENTS WHICH MAY OCCUR IN THE COURSE OF BLOCKING THE SPLANCHNIC NERVES AND THE FIRST LUMBAR SYMPATHETIC GANGLION

Normally, anesthetic block of the splanchnic nerves or of these nerves and the first lumbar sympathetic ganglion is well tolerated by patients. Some patients, however, especially those that have low arterial blood pressure, may feel after the injection of the anesthetic solution a temporary and slight sensation of dizziness or faintness, sometimes accompanied with a subjective visual disturbance. These sensations are probably caused by cerebral ischemia at the same time that the blood pressure becomes lower. This ischemia is probably due to visceral vasodilatation in the field of the innervation of the splanchnic nerves blocked by the anesthetic solution. To avoid these disturbances, which may appear a few minutes after anesthetic block of the splanchnic nerves, the patient is always placed in the dorsal decubitus, with head low. The sensations disappear completely, without damage to the patient, one hour or less after the anesthetic injection.

During the block of the splanchnic nerves, the tip of the needle may sometimes perforate a deep vessel and blood may appear in the hub of the needle. It is important to avoid injection of the anesthetic solution into a vessel. To be sure that the injection is not intravascular, it is necessary to aspirate with the syringe before the injection is made and during the injection if the direction of the needle changed with the maneuvers of the injection. In a few instances, I have observed that when the needle was introduced to block the left splanchnic nerves the patient sometimes felt pain at the level of the heart. This pain disappeared after the injection of the anesthetic solution. Investigations that I have carried out on patients on whom I have performed splanchnicectomy while they were under local anesthesia showed that mechanical stimulation of the left splanchnic nerves may produce pain at the level of the heart. Anesthesia of these nerves during the mechanical stimulation immediately abolished the precordial pain. These facts led me to believe that pain at the level of the heart produced by the introduction of the needle to reach the left splanchnic nerves is probably caused by the stimulation of the left splanchnic nerves.

During a period of eight years of investigation with single or repeated anesthetic blocks of the splanchnic nerves, I have observed only one serious complication, in a patient with gastric ulcer who was being treated by repeated anesthetic blocks of the splanchnic nerves. Once, when the needle was introduced into the left side and when the injection of the anesthetic solution started, the patient immediately had a convulsion, lost consciousness and stopped breathing. Oxygen was administered, and respiration was restored by artificial means. A few minutes later the patient began to breathe, and twenty minutes

later he became conscious. It was then seen that the right upper and lower extremities were paralyzed. Twenty-four hours later paralysis had not improved. In attempting to interpret this accident, which I had never seen before, I began to think that it was probably the result of a cerebral vascular spasm, caused by the mechanical stimulation of the left splanchnic nerves. In accordance with this hypothesis, I performed an anesthetic block of the left middle and stellate ganglions, which was followed by a favorable influence on the paralysis of the extremities. Two days later another block of the same ganglions was performed. One week after the accident, the patient had recovered and the paralysis of the extremities of the right side had disappeared.

During my studies in this field, with more than one thousand anesthetic blocks of the splanchnic nerves, I never saw a repetition of this accident. The favorable influence of the anesthetic block of the middle cervical and stellate ganglions and the disappearance of the paralysis suggest that in this case the mechanism of the paralysis was probably a reflex spasm of the vessels of the brain caused by the mechanical stimulation of the left splanchnic nerves.

CLINICAL INDICATIONS

The clinical problems related to the splanchnic nerves have been investigated more intensively in the last years than formerly and have particularly interested the surgeons. The results already obtained are encouraging. In a complex field such as the splanchnic nerves and the solar plexus, one must proceed with caution to find the right procedure and to learn to interpret the results of clinical effects through action on these nerves. From the studies carried on up to this time, it is deduced that the single or the repeated anesthetic block of the splanchnic nerves seems to be a useful method by which to study the effects of temporary interruption of the splanchnic nerves alone or associated with the sympathetic chain at the level of the first lumbar ganglion.

I. *Anesthetic Block of the Splanchnic Nerves.*—1. In Cardiospasm: The cardia is innervated by the vagus and sympathetic nerves. Motor control of the cardia and lower part of the esophagus is effected by local automatism and reflexes through Auerbach's plexus and by long reflexes through the vagi efferents and through the splanchnic efferents (Carlson, Boyd and Pearcy [29]). Carlson [30] observed in the cat that the splanchnic nerves, like the vagi, contain both motor and inhibitory

29. Carlson, A. J.; Boyd, T. E., and Pearcy, J. F.: Studies on the Visceral Sensory Nervous System: XIV. The Reflex Control of the Cardia and Lower Esophagus, Arch. Int. Med. **30**:409 (Oct.) 1922.

30. Carlson, A. J.; Boyd, T. E., and Pearcy, J. F.: Studies in the Visceral Sensory Nervous System: XIII. The Innervation of the Cardia and the Lower End of the Esophagus in Mammals, Am. J. Physiol. **61**:14, 1922.

efferent fibers to the cardia and lower part of the esophagus. Carlson and co-workers [29] demonstrated in the cat and dog that the inhibition reflex seems to be evoked more readily through the vagal efferent and the motor reflex through the splanchnic efferent fibers. Cardiac reflexes initiated by the splanchnic afferent fibers are predominantly motor.

According to Hurst and Rake,[31] four abnormal conditions may affect the nervous control of the cardiac sphincter: vagus hyperactivity or hypoactivity and sympathetic hyperactivity or hypoactivity. Absence of vagus impulses, resulting in achalasia of the cardia and overaction of the sympathetic impulses producing cardiospasm, are the two factors that probably play the most important part in dysfunction of the cardiac sphincter. Cardiospasm may occur as a reflex result of acute inflammation or carcinoma of the lower portion of the esophagus, peptic ulcer and possibly duodenal ulcer or gallbladder disease (Kuntz [32]). In spasm of the cardiac sphincter, Pottenger [33] pointed out the presence of sympathetic reflexes. In the production of these reflexes, both afferent and efferent impulses course over the sympathetic system.

The conception of treatment of cardiospasm through action on the sympathetic innervation was tested in patients after it was demonstrated that in the bilaterally vagotomized cat (Knight [34]) or monkey (Ferguson [35]) the presence of cardiospasm can be relieved by section of the sympathetic nerves that supply the sphincter. In 2 cases of achalasia with cardiospasm and in 1 case of hypertrophic stenosis of the cardia, Knight [36] performed resection of the left gastric artery with its surrounding nerves that supply the cardiac sphincter. The first 2 patients were improved by sympathectomy, but the third patient was not completely relieved. Meade [37] reported 1 case of sympathectomy, and Ochsner and DeBakey [38] reviewed the literature and collected 19 cases of achalasia in which sympathectomy was done. There was 1 death;

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31. Hurst, A. F., and Rake, G. W.: Achalasia of the Cardia, Quart. J. Med. **23**:491, 1930.

32. Kuntz, A.: Autonomic Nervous System, ed. 2, Philadelphia, Lea & Febiger, 1934, p. 530.

33. Pottenger, F. M.: Symptoms of Visceral Disease, St. Louis, C. V. Mosby Company, 1944, p. 253.

34. Knight, G. C.: The Relation of the Extrinsic Nerves to the Functional Activity of the Esophagus, Brit. J. Surg. **22**:155, 1934.

35. Ferguson, J. H.: Effects of Vagotomy on the Gastric Functions of Monkeys, Surg., Gynec. & Obst. **62**:689, 1936.

36. Knight, G. C.: Sympathectomy in the Treatment of Achalasia of the Cardia, Brit. J. Surg. **22**:864, 1935.

37. Meade, H. S.: A Case of Sympathectomy in the Treatment of Achalasia of the Cardia, Irish J. M. Sc., 1939, p. 130.

38. Ochsner, A., and DeBakey, M.: Surgical Considerations of Achalasia: Review of the Literature and Report of Three Cases, Arch. Surg. **41**:1146 (Nov.) 1940.

recurrence took place in 4 cases, and improvement was only partial in 4 patients. In a patient with cardiospasm and pain, Craig, Moersch and Vinson [39] performed bilateral resection of the cervicothoracic sympathetic trunk and ganglions. After the operation the patient was completely relieved of dysphagia and of pain, and she has had no further paroxysmal attacks of the acute type of pain. White and Smithwick [40] pointed out that the results on the whole are not impressive and that further experience is essential. Since previous investigations had put in evidence the probable role of the sympathetic system in the mechanism of cardiospasm, I have tried to analyze its influence through interruption of the splanchnic nerves by anesthetic block. Some facts were observed that can help in the study of the role of sympathetic innervation in cardiospasm.

The first patient to be treated for cardiospasm had also clinical and radiologic signs of megaesophagus and duodenal ulcer. During two weeks, six anesthetic blocks of the right and left splanchnic nerves were performed and the cardiospasm disappeared. Ten more anesthetic blocks were carried out, and one and a half months after the beginning of this treatment the clinical signs of duodenal ulcer and cardiospasm were completely absent. Three years after this treatment the patient remained in good condition. During this period there was no return of duodenal ulcer or cardiospasm.

In a young patient with cardiospasm and megaesophagus, without evident lesions of the stomach or esophagus, repeated anesthetic blocks of the left splanchnic nerves were performed. Five anesthetic blocks in two weeks completely relieved the cardiospasm. For eight months he remained in good condition, but after this period a new crisis of cardiospasm appeared. A new series of eight anesthetic blocks of the left splanchnic nerves was performed, and the cardiospasm was relieved for the second time. At the present time this patient is under observation to see if a new crisis of cardiospasm appears.

A third patient with cardiospasm and megaesophagus was studied. Four weeks after the beginning of his esophagic disturbance there was a narrowing at the level of the cardia that allowed only the passage of fluids. In this patient four anesthetic blocks of the left splanchnic nerves, performed in two days, were not followed by any change in the permeability of the cardia. This patient died of pneumonia two weeks later. Anatomic study of the cardia showed a narrow stenosis caused by scar tissue probably due to healed cardiac ulcer.

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39. Craig, W. M.; Moersch, H. J., and Vinson, P. P.: Treatment of Intractable Cardiospasm by Bilateral Cervicothoracic Sympathetic Ganglionectomy: Report of a Case, Proc. Staff Meet., Mayo Clin. **9**:749, 1934.

40. White, J. C., and Smithwick, R. H.: Autonomic Nervous System, New York, The Macmillan Company, 1944, p. 356.

In a patient with inoperable cancer of the lower portion of the esophagus there was cardiospasm associated with the cancer. Three anesthetic blocks of the left splanchnic nerves brought relief from the cardiospasm during the days of treatment. After splanchnicectomy on the left side was performed, the patient remained improved of her cardiospasm for about one month and then died of internal hemorrhage.

The facts observed in the cases of these patients suggest that repeated anesthetic blocks of the left splanchnic nerves may be useful for relief of cardiospasm, at least in certain cases, also as a method of differential diagnosis between cardiospasm and organic stenosis and as a preoperative test before splanchnicectomy.

2. In Visceral Pain: At the present time it is known that afferent impulses from internal viscera innervated by sympathetic fibers run centrad by afferent spinal neurons which accompany the sympathetic visceral nerves (Pottenger [33]). The visceral afferent pathways enter the spinal cord in the posterior roots, and their axons run to the peripheral plexuses in the visceral nerves. The splanchnic nerves carry the afferent sensory pathways that innervate the stomach, small intestine, liver, gallbladder and cystic biliary ducts (Davis, Hart and Crain [41]). In pain of the stomach, Head [42] and Läwen have demonstrated that the sensory afferent pathways enter the spinal cord in the sixth, seventh, eighth and ninth thoracic segments. According to Gubergritz and Istschenko,[43] the painful afferent impulses from the kidneys are conducted through the renal plexus and reach the spinal cord through the splanchnic nerves. Investigations made on patients have confirmed the presence of afferent sensory pathways in the splanchnic nerves conducting painful sensations. Mechanical stimulation of the splanchnic nerves performed in patients by Adson [44] and Leriche [45] provoked pain that was relieved by anesthetic block of the splanchnic nerves. Bentley and Smithwick [46] observed that epigastric pain produced by insufflation of a balloon at a certain pressure in the upper jejunum disappeared to the

41. Davis, L.; Hart, J. T., and Crain, R. C.: The Pathways for Visceral Afferent Impulses Within the Spinal Cord: Experimental Dilatation of the Biliary Ducts, Surg., Gynec. & Obst. **48**:647, 1929.

42. Head, H.: On Disturbances of Sensation with Special Reference to the Pain of Visceral Disease, Brain **16**:1, 1893.

43. Gubergritz, M. M., and Istschenko, I. N.: Zur Frage der Entstehung der Schmerzempfindungen in den Nieren, Ztschr. f. d. ges. exper. Med. **52**:619, 1926.

44. Adson, A. W.: Splanchnic Pain, Proc. Staff Meet., Mayo Clin. **10**:623, 1935.

45. Leriche, R.: Des douleurs provoquées par l'excitation du bout central des grands splanchniques (douleurs cardiaques, douleurs pulmonaires) au cours des splanchnicotomies, Presse méd. **45**:971, 1937.

46. Bentley, F. H., and Smithwick, R. H.: Visceral Pain Produced by Balloon Distention of the Jejunum, Lancet **2**:389, 1940.

right of the midline after the performance of splanchnicectomy on the right side and lumbar sympathectomy. The same degree of distention was not felt after bilateral splanchnicectomy. Low abdominal pain from the lower part of the intestinal tract was relieved in a patient by splanchnicectomy on the right side and resection of the upper lumbar sympathetic ganglions (White and Smithwick [40]). In patients with chronic biliary pain that was intractable to surgical operation, splanchnicectomy was tried, with successful results (Craig [47] and White and Smithwick [40]). From these facts, it is deduced that the fibers carrying painful sensations from the organs innervated by the solar plexus pass through the splanchnic nerves to reach the spinal cord. On the other hand, up to this time it had not been demonstrated that sensory fibers carrying painful sensations from the viscera innervated by the solar plexus pass through the vagus nerves. Then, if the splanchnic nerves are interrupted temporarily by anesthetic block or definitively by splanchnicectomy, visceral pain is relieved in the area innervated by the sensory fibers of these nerves. Knowing this fact, I have been employing anesthetic block as a method of diagnosis or of treatment and as a preoperative test in the indication of splanchnicectomy to relieve visceral pain. As a method of diagnosis, it has been useful to differentiate deep visceral pain from the parietal pain originating at the level of the abdominal wall as it is seen in parietal pain caused by epigastric hernia or in other types of parietal pain. The information obtained by anesthetic block of the splanchnic nerves may be controlled by infiltration with an anesthetic solution of the area of the abdominal wall whence arises the painful stimulation. Anesthetic block of the splanchnic nerves was also important to determine in every case the topography of the sensory fibers that carry the painful visceral sensations.

In cases of inoperable cancer of the stomach accompanied with pain,[48] I have observed that repeated anesthetic blocks of the splanchnic nerves were followed by relief of visceral pain only during one to one and a half hours. However, in a few cases I have observed that anesthetic blocks were followed by relief from pain during a period varying from two hours to few days. In the cases in which relief of pain corresponds only to the time of anesthesia of the splanchnic nerves, there is probably stimulation of the sympathetic innervation by the neoplastic tissue. In the cases in which anesthetic block was followed by relief of pain during several hours or days, the physiologic interruption probably exerts its influence by relieving the vascular spasm that may be a cause of the

47. Craig, W. M.: Surgical Approach to and Resection of the Splanchnic Nerves for Relief of Hypertension and Abdominal Pain, West. J. Surg. **42**:146, 1934.

48. de Sousa Pereira, A.: A cirurgia simpatica no tratamento das algias dos cancros inoperaveis do estomago, Med. contemp. **60**:80, 1941.

visceral pain. In the surgical treatment of pain in inoperable cancer of the stomach and the pancreas, I have employed routinely the anesthetic block of the splanchnic nerves to determine before the operation whether it was through the left, through the right or through both splanchnic nerves that the afferent sensory fibers carrying the visceral painful sensations ran. By the use of this method, it was possible to obtain an accurate indication regarding the topography of the afferent painful pathways that pass through the splanchnic nerves and the probable result of unilateral or bilateral splanchnicectomy as treatment of visceral pain.

3. In Peptic Ulcer: Much evidence has been collected by several authors, particularly during the last years, that points out the probable role of the autonomic nervous system in the pathogenesis of peptic ulcer. It was demonstrated that in certain brain tumors and following hypothalamic lesions caused by operations on the brain (Cushing [49] and Grant [50]) gastroduodenal erosions, perforations or ulcers may occur. On the other hand, the role of emotions and of psychosomatic disturbances in the pathogenesis of peptic ulcer has been emphasized by several authors (Cushing,[49] Robinson,[51] Bolen,[52] Winkelstein and Rothschild,[53] Wolf [54] and Wolff [55]).

For several years Dragstedt [56] has been studying the problem of the pathogenesis of peptic ulcer. In a complete and interesting work published in 1942, he emphasized his belief that "the problem of the cause of gastric and duodenal ulcer is a part of the more general question of the resistance of the gastrointestinal tract to the digestive action of its own secretions." In the same year, Palmer [57] pointed out that the problem of peptic ulcer in man is one of tissue resistance versus acid attack. The point of view of these authors seems to be in exact accord with the

49. Cushing, H.: Peptic Ulcers and Interbrain, Surg., Gynec. & Obst. **55**:1, 1932.

50. Grant, F. C.: Brain Lesions and Duodenal Ulcer: Report of Two Cases, Ann. Surg. **101**:156, 1935.

51. Robinson, S. C.: Role of Emotions in Gastroduodenal Ulcers, Illinois M. J. **71**:338, 1937.

52. Bolen, H. L.: The Emotional Factor in Peptic Ulcer, Rev. Gastroenterol. **10**:187, 1943.

53. Winkelstein, A., and Rothschild, L.: Some Clinical Studies on the Psychosomatic Background of Peptic Ulcer, Am. J. Digest. Dis. **10**:99, 1943.

54. Wolf, S., and Wolff, H. G.: Human Gastric Function, New York, Oxford University Press, 1943.

55. Wolff, H. G.: Emotions and Gastric Function, Science **98**:481, 1943.

56. Dragstedt, L. R.: Pathogenesis of Gastroduodenal Ulcer, Arch. Surg. **44**: 438 (March) 1942.

57. Palmer, W. L.: Peptic Ulcer and Gastric Secretion, Arch. Surg. **44**:452 (March) 1942.

present interpretation of the problem of peptic ulcer and includes the two factors which must be considered when the pathogenic mechanism of peptic ulcer is analyzed: (1 the gastroduodenal wall and its power of resistance versus (2) the peptic activity of the gastric secretion. Probably the mechanism of peptic ulcer is variable from case to case, and in every one the principal role is played by one or both of these factors.

The vagal neurogenic conception of gastric hypersecretion was accepted by Dragstedt and Owens,[58] who in 2 cases of duodenal ulcer performed supradiaphragmatic section of the vagus nerves. From the study of the 2 patients, the authors concluded that the data obtained indicate that the excessive night secretion of gastric juice in patients with ulcer is probably neurogenic in origin and may be greatly reduced by supradiaphragmatic section of the vagus nerves.

In 1945 Dragstedt and Schafer[59] reported the results that they had obtained in 13 patients with duodenal ulcer, in 1 patient with gastric ulcer and in 1 with gastrojejunal ulcer following supradiaphragmatic division of the vagus nerves. The patient with gastric ulcer has been apparently cured of his disease, and all but 1 of the patients with duodenal ulcer have been greatly improved or cured, although 3 patients have required gastroenterostomy because of persistence of obstructive symptoms. The patient with gastrojejunal ulcer, although improved, has been continued on medical management. In these patients the excessive continuous night secretion of gastric juice has been greatly reduced by the vagus section, a fact which, according to Dragstedt and Schafer, favors the conception that this abnormality is neurogenic in origin and the view that gastroduodenal ulcer is a psychosomatic disease. I was especially interested in the study of the first factor, the resistance of the gastroduodenal wall. I started from the hypothesis that if the decreased resistance of the gastroduodenal wall favors the genesis of peptic ulcer the progressive increase of resistance of the same wall must speed the healing of an existing ulcer. To increase locally the resistance of the gastroduodenal wall, I exerted action on the blood supply by temporary or definitive interruption of the vasomotor innervation of the stomach and duodenum. By repeated anesthetic blocks or by splanchnicectomy, I interrupted the splanchnic nerves in patients with ulcers, and a favorable result was observed.[60]

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58. Dragstedt, L. R., and Owens, F. M.: Supradiaphragmatic Section of the Vagus Nerves in the Treatment of Duodenal Ulcer, Proc. Soc. Exper. Biol. & Med. **53**:152, 1943.

59. Dragstedt, L. R., and Schafer, P. W.: Removal of the Vagus Innervation of the Stomach in Gastroduodenal Ulcer, Surgery **17**:742, 1945.

60. de Sousa Pereira, A.: O tratamento das úlceras gastro-duodenais pelos bloqueios anestésicos dos nervos esplancnicos, Portugal méd., 1942, no. 1, p. 1.

At the same time, I have approached this problem on an experimental basis.[61] In a series of 30 dogs under general ether anesthesia and after previous gastrotomy, my co-workers and I [61] have made wounds in the gastric mucosa. The diameters of those wounds varied—1.50 cm. in 3 dogs, 2 cm. in 18 dogs and 5 cm. in the last 9 dogs. The wounds embraced the full thickness of the gastric mucosa. In 20 of these animals the wound was made in the antrum, in 7 in the region of the fundus and in 3 at the pylorus. In 13 dogs only resection of the gastric mucosa in the regions just mentioned was done. In 16 dogs, after the gastric wound was made, unilateral splanchnicectomy was performed, and in 1 dog bilateral splanchnicectomy was done. All the animals were kept under observation for periods ranging from eight to thirty days and were then killed. It was observed that there was a remarkable

Fig. 11.—Wound of the gastric mucosa made in the region of the antrum. In the figure on the left (dog 1000), the wound is ten days old. In that on the right (dog 1002), the wound was followed by right splanchnicectomy and is nine days old.

acceleration of healing in the animals in which the wound of the gastric mucosa had been followed by splanchnicectomy (fig. 11).

The results of these experimental investigations are in accordance with the facts that I have observed in patients. In 3 cases of ulcer of the lesser curvature, I have performed repeated (eight to twenty) anesthetic blocks of the splanchnic nerves. These blocks were made during a period of nineteen to twenty-two days; and it was demonstrated by roentgenologic examination that the ulcer cavity had disappeared

61. de Sousa Pereira, A.; Rodrigues, A., and Carvalho, R.: A influência da interrupção dos nervos esplâncnicos sôbre a cicatrização das feridas gástricas, Portugal méd., 1941, no. 1, p. 1.

during that period (fig. 12). Gastrectomy was then performed, and the gastric specimen was studied. The anatomic and histologic study in every case confirmed the advanced phase of healing and organization of the scar tissue in the spot where an ulcer cavity had been shown by roentgenogram before treatment (fig. 13).

Section of the splanchnic nerves causes interruption of the vasomotor fibers that pass through them. I tried to determine the influence of splanchnicectomy on the arterial collateral circulation of the stomach. In a series of 10 dogs the vessels at the level of the greater curvature were interrupted by ligation. In 5 dogs, interruption of these vessels was performed and the animals were kept under observation. In 5 other animals, interruption of the vessels of the greater curvature was followed by splanchnicectomy. These animals have been studied

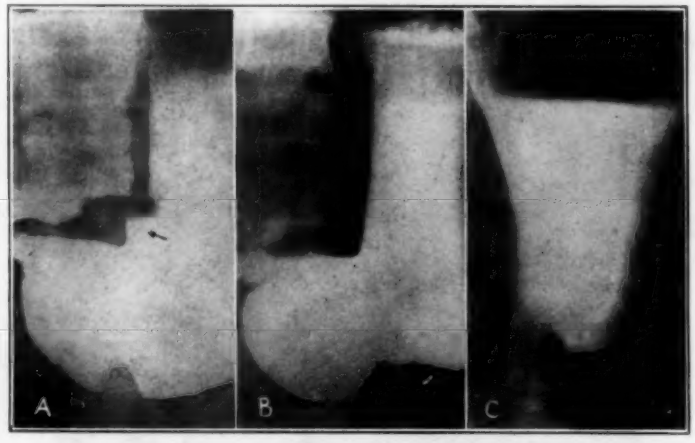

Fig. 12.—Ulcer of the lesser curvature in a patient (F. R.) 31 years old. *A*, observation on Oct. 18, 1943, before treatment. *B*, observation on November 12, after twenty splanchnic nerve blocks made during twenty-two days. *C*, observation on December 3. Gastrectomy was done on November 15.

by arteriography one to four weeks after the operation. It was observed that in animals in which only the interruption of the vessels of the greater curvature was performed the development of the arterial collateral circulation was uniform through the entire area of the gastric wall (fig. 14), but in 5 dogs in which ligation of the vessels of the greater curvature was followed by splanchnicectomy there was a greater development in the arterial collateral circulation. In the two thirds of the stomach corresponding to the side of the interruption of the splanchnic nerves (fig. 15), the increase of collateral arterial supply was evident.

In April 1939 I began studying the influence of repeated anesthetic blocks of the splanchnic nerves in the treatment of peptic ulcer.[60] From that time until May 1944 I had studied carefully and closely a series of 18 patients with peptic ulcer: 8 with ulcer of the lesser curvature; 4

with pyloric or justapyloric ulcer; 2 with duodenal ulcer and 4 with multiple ulcers. In 17 of these patients it was possible by repeated anesthetic blocks of the splanchnic nerves, performed during periods varying from one to two months, to obtain relief of clinical signs and complete healing of the ulcer demonstrated in every case by the roentgenologic examination (figs. 16, 17 and 18). In 1 case in which there was a penetrating ulcer of the lesser curvature, no healing of the ulcer

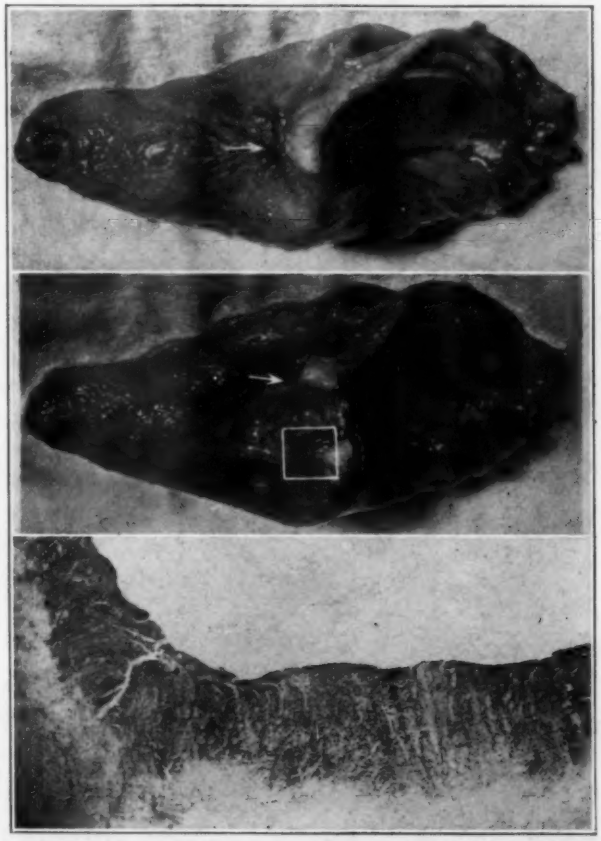

Fig. 13.—Ulcer of the lesser curvature in a patient (F. R.) 31 years old. Gastrectomy specimen obtained after the treatment by twenty splanchnic nerve blocks made during twenty-two days. Histologic section of the scar of the gastric ulcer.

was obtained and only temporary relief of the epigastric pains. In these patients the periods of observation after the treatment ranged from six months to five years. In 1 case of ulcer of the lesser curvature in this series a recurrent lesion was observed a year later in a different point of the lesser curvature. A new series of anesthetic blocks of the splanchnic nerves quickly relieved the clinical signs and speeded the

healing of the ulcer. Also in 1 case of duodenal ulcer, a recurrent lesion was observed three years after the treatment. Repeated anesthetic blocks were performed, and the ulcer healed. Fifteen months later the patient still was in good condition. The pyloric and justapyloric ulcers

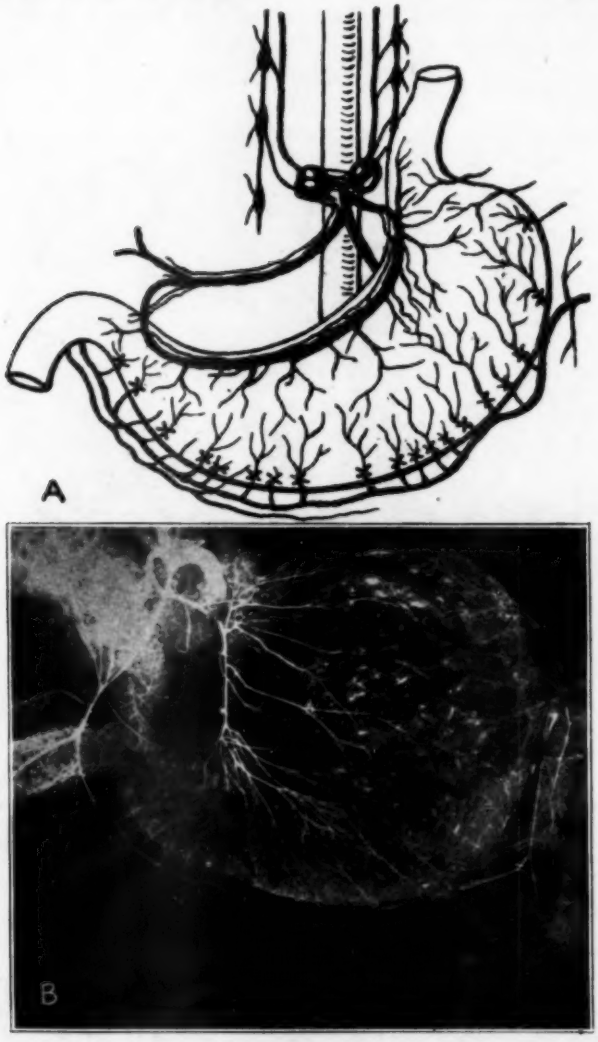

Fig. 14 (experiment 1, dog 988).—*A*, ligation of the vessels of the greater curvature of the stomach. *B*, arteriography done twenty-three days after operation. The collateral circulation developed between the arteries of the two curvatures is practically the same in the whole gastric area.

healed, but a pyloric stenosis appeared in 3 patients that needed surgical operation to relieve the stenosis.

The study of these cases showed that pain is quickly relieved and that peptic ulcer can be completely healed by increase of the arterial

blood supply of the gastroduodenal wall by repeated anesthetic blocks of the splanchnic nerves. Only in 1 patient of this series with a penetrating ulcer of the lesser curvature was this result not obtained. After I observed the recurrence of ulcer in 2 cases, I began, two years ago, to perform interruption of the splanchnic nerves by splanchnicectomy

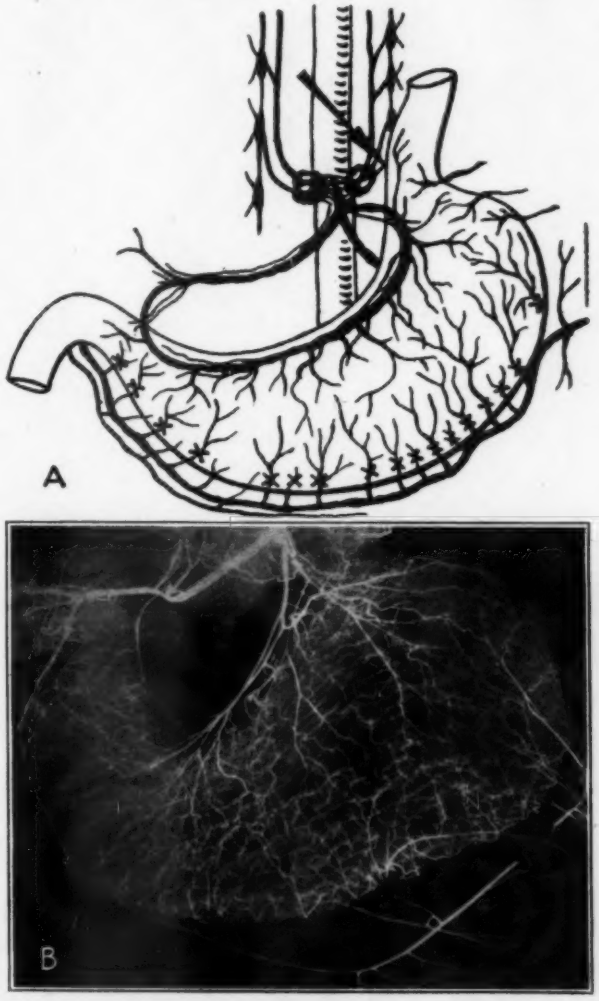

Fig. 15 (experiment 11, dog 981).—*A*, ligation of the vessels of the greater curvature followed by left splanchnicectomy. *B*, arteriography, twenty days after operation, shows a greater development of the collateral arterial circulation in the left two thirds of the stomach.

in cases of peptic ulcer. All experimental and clinical data of my investigations regarding the problem of peptic ulcer and autonomic nervous system will soon be published.

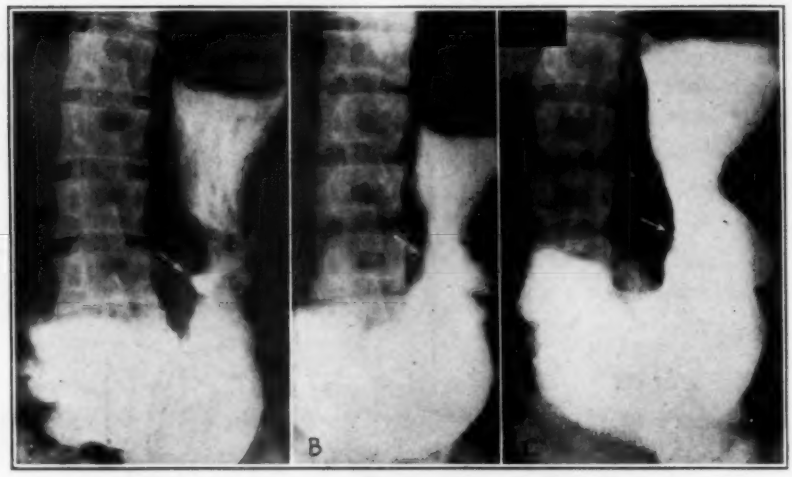

Fig. 16.—Ulcer of the lesser curvature in a patient (I. C.) 45 years old. *A*, observation on Oct. 23, 1943, before the treatment. *B*, observation on December 2, after twenty splanchnic nerve blocks practiced during twenty-four days. *C*, observation December 12, after twenty-six splanchnic nerve blocks practiced during thirty-four days.

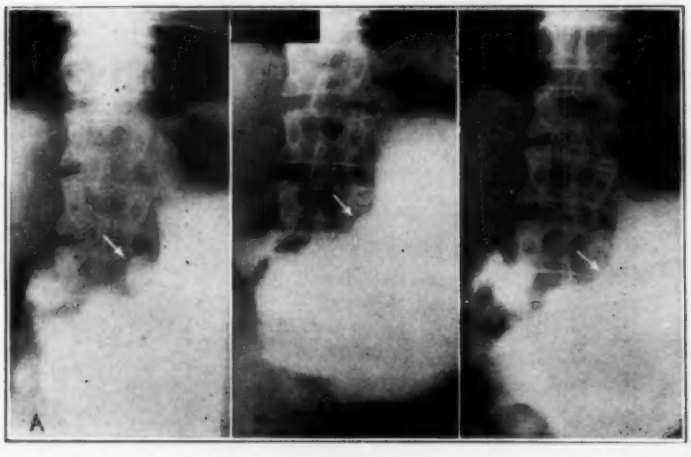

Fig. 17.—Ulcer of the lesser curvature in a patient (M. P. B.) 49 years old. *A*, observation on Nov. 8, 1943, before treatment. *B*, observation on December 2, after sixteen splanchnic nerve blocks during twenty-two days. *C*, observation on December 17, after thirty splanchnic nerve blocks practiced during thirty-seven days.

II. *Anesthetic Block of the Splanchnic Nerves and First Lumbar Ganglion.*— 4. In Paralytic Ileus: The intestine is innervated by both parasympathetic and sympathetic nerves, the first activating and the latter exerting a tonic inhibitory influence (Pottenger [33]). Excitation of the splanchnic nerves causes inhibition of the intestine, affecting both longitudinal and circular coats (Bayliss and Starling [62]). Interruption of these nerves favors the intestinal motor activity of the opposing parasympathetic system. However, both the sympathetic nerves and the vagus nerves may conduct both motor and inhibitory impulses to

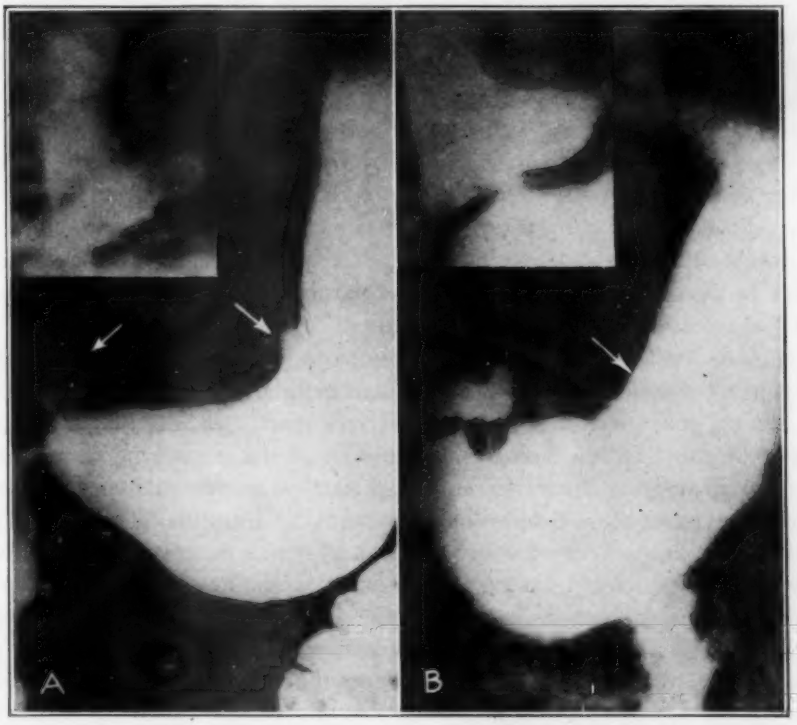

Fig. 18.—Multiple ulcers (ulcer of the lesser curvature and duodenal ulcer) in a patient (L. P.) 47 years old. *A*, observation on Oct. 9, 1942, before the treatment. *B*, observation on December 22, after thirty-one splanchnic nerve blocks during forty-nine days.

the gastrointestinal musculature. Bilateral section of the splanchnic nerves results in increased tone and augmented peristaltic activity of the gastrointestinal musculature (Kuntz [32]). This physiologic knowledge of the innervation of the intestine offers the basis for the treatment of paralytic ileus by anesthetic block of the intestinal sympathetic innervation.

62. Bayliss, W. B., and Starling, E. H.: The Movements and Innervation of the Small Intestine, J. Physiol. **24**:99, 1899.

Several clinical reports have appeared since Wagner,[63] in 1922, made known the favorable results of spinal anesthesia in the treatment of ileus. Duval [64] reported that evacuation of the intestine was brought about in 68 per cent of the cases of adynamic, paralytic and spasmodic ileus. Markowitz and Campbell,[65] in experimental investigations, observed that the reflex paralysis of the bowel movements induced in dogs by laparotomy or by the intraperitoneal injection of iodine or by severe intra-abdominal traumatism is promptly abolished by spinal anesthesia induced by procaine hydrochloride. Ochsner, Gage and Cutting [66] suggested, in 1928, the treatment of ileus by the mere blocking of the splanchnic nerves. They performed twenty-five experiments on dogs to determine the effect of splanchnic anesthesia in ileus. They stated the belief that physiologic, chemical and paralytic ileus or mechanical ileus following the relief of an obstruction may be satisfactorily treated in the experimental animal by block of the splanchnic nerves. Rosenstein and Köhler [67] observed that in rabbits peristalsis was produced following the injection of large amounts of procaine hydrochloride solution into the splanchnic area. These experimental reports are favorable to the conception of treatment of paralytic ileus by anesthetic block of the intestinal sympathetic innervation.

Since 1938 [68] I have routinely employed bilateral anesthetic block of the splanchnic nerves and first lumbar ganglion in every case in which a clinical diagnosis of paralytic ileus was made. It was observed that in some cases the physiologic interruption of the splanchnic nerves and first lumbar ganglion increased the peristaltic movements of the intestine and was followed after the treatment by intestinal discharge. In other cases, the effect was only partial and it was necessary to add other therapeutic methods to complete the effect of the anesthetic block. In a few cases it had no influence on the intestinal function. When the intestinal effect following the first anesthetic block was only partial or absent and it was necessary to prolong the action of the interruption

63. Wagner, G. A.: Zur Behandlung des Ileus mit Lumbalanästhesie, Arch. f. Gynäk. **117**:336, 1922.

64. Duval, P.: La rachianesthésie dans l'occlusion intestinale, Bull. et mém. Soc. nat. de chir. **53**:477, 1927.

65. Markowitz, J., and Campbell, W. R.: The Relief of Experimental Ileus by Spinal Anesthesia, Am. J. Physiol. **81**:101, 1927.

66. Ochsner, A.; Gage, I. M., and Cutting, R. A.: Treatment of Ileus by Splanchnic Anesthesia: Preliminary Report of Experimental Study, J. A. M. A. **90**:1847 (June 9) 1928.

67. Rosenstein, P., and Köhler, H.: Therapeutische Versuche zur Bekampfung der Darmlaemung durch Umspritzung des Ganglion coelicum, Med. Klin. **22**:530, 1926.

68. de Sousa Pereira, A.: A infiltracao novocainica dos nervos esplancnicos na terapeutica da oclusao intestinal, Med. contemp., April 10, 1938.

of the sympathetic innervation of the intestine, a second anesthetic block was performed after the effect of the first block had disappeared. If it was not followed by a favorable result when associated with other therapeutic methods a laparotomy was performed. In a few instances in which the anesthetic blocks of the splanchnic nerves had given unsuccessful results, a mechanical cause was found when the laparotomy was practiced. In the cases of paralytic ileus in which the anesthetic block was successful, daily repeated anesthetic blocks were performed at the level of the splanchnic nerves and first lumbar ganglion until complete reestablishment of the intestinal function was obtained. In all cases of ileus in which the patients were treated by anesthetic blocks, the general and local condition of the patient and especially the arterial blood pressure were carefully evaluated before and during the treatment because interruption of the splanchnic nerves is followed by hypotension.

5. In Megacolon: Both sympathetic and parasympathetic nerves innervate the colon. Schmidt,[69] experimenting on cats and dogs, observed that the vagus nerves supply the ascending and transverse portions of the colon whereas the sacral fibers are distributed to the entire colon. These results confirm the physiologic observations of Carlson [70] on the dog, in which irritation of the sacral nerves produces a motor effect on the circular and longitudinal musculature over the entire bowel. On the other hand, it is admitted at the present time (Pottenger [38]) that the sympathetic pathways for the colon come through the lower thoracic and upper lumbar segments of the cord and reach the colon through the superior and inferior mesenteric ganglions.

Since Wade and Royle [71] have observed improvement in bowel activity following lumbar ramisection in cases of spastic paraplegia, different types of sympathetic operations have been performed in the treatment of megacolon. Judd and Adson [72] obtained satisfactory results from lumbar sympathetic ganglionectomy and ramisectomy in the treatment of 2 patients suffering from congenital idiopathic dilatation of the colon. Rankin and Learmonth [73] performed resection of the inferior

69. Schmidt, C. A.: Distribution of Vagus and Sacral Nerves to the Large Intestine, Proc. Soc. Exper. Biol. & Med. **30**:739, 1933.

70. Carlson, A. J.: The Extrinsic Nervous Control of the Large Bowel, J. A. M. A. **94**:78 (Jan. 11) 1930.

71. Wade, R. B., and Royle, N. D.: The Operative Treatment of Hirschsprung's Disease: New Method with Explanation of the Technic and Results, M. J. Australia **1**:137, 1927.

72. Judd, E. S., and Adson, A. W.: Lumbar Sympathectomy and Ramisectomy for Congenital Idiopathic Dilatation of the Colon, Ann. Surg. **88**:479, 1928.

73. Rankin, F. W., and Learmonth, J. R.: The Present Status of the Treatment of Hirschsprung's Disease, Am. J. Surg. **15**:219, 1932.

mesenteric and presacral nerves in 8 cases, with return of the function of the colon, although tardily in some cases. Barrington-Ward [74] carried out the same operation in 2 cases, with good results. Adson [75] and Leriche [76] in the same year introduced splanchnicectomy alone or accompanied with lumbar sympathectomy in the treatment of megacolon. Leriche and co-workers [77] obtained favorable results in patients in whom this type of sympathetic denervation of the colon was performed.

Spinal anesthesia as a test of sympathetic overactivity was introduced by Scott and Morton.[78] In 2 clinical cases of Hirschsprung's disease they have demonstrated the immediate augmentation in motor activity of the large bowel on the induction of spinal anesthesia. They have proposed the adoption of this procedure to ascertain in any individual case of megacolon how effective operative interruption of its sympathetic innervation will prove to be.

The difficulties that I have encountered in the indication for splanchnicectomy and lumbar sympathectomy in cases of Hirschsprung's disease led me to study, as a preoperative test, the physiologic interruption of the splanchnic nerves and first lumbar ganglion by repeated anesthetic blocks. Three cases of megacolon were studied by this method. Daily repeated anesthetic blocks of the splanchnic nerves and first lumbar ganglion were performed alternately in the right and left sides during a period of two weeks. These patients were studied clinically and by roentgenographic examination before, during and after the treatment in similar technical conditions in order that the results might be compared. This study showed in these cases that repeated anesthetic blocks were followed by functional and morphologic improvement of the colon. Splanchnicectomy and resection of the first lumbar sympathetic ganglion were then performed. These 3 patients with megacolon were operated on according to the indication of this test. The immediate results obtained and those which followed during a period of observation ranging from five months to two years have been encouraging. The small number of cases, however, and the short period of observation do not permit a definitive conclusion.

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74. Barrington-Ward, L. E.: Abdominal Sympathectomy for Hirschsprung's Disease, Proc. Roy. Soc. Med. **25**:1221, 1932.

75. Adson, A. W.: Hirschsprung's Disease: Indications for and Results Obtained by Sympathectomy, Surgery **1**:859, 1937.

76. Leriche, R.: De la section des splanchniques dans le mégacolon non compliqué avec ou sans dolichocolon, Presse méd. **45**:1851, 1937.

77. Leriche, R.; de Sousa Pereria, A., and DeBakey, M.: La resection des nerfs splanchniques: Technique et resultats de quelques observations de thromboangeite oblitérante, hypertension paroxystique, megacolon et dolichocolon, Med. contemp. no. 27, 1937.

78. Scott, J. M., and Morton, J. J.: Sympathetic Inhibition of the Large Intestine in Hirschsprung's Disease, J. Clin. Investigation **9**:247, 1930.

6. In Hypertension: Although the cause and mechanism of essential hypertension remain unknown, great progress has been made during recent years in the surgical treatment of this disease. According to White and Smithwick,[40] the most important factors in this disease seem to be primary vascular disease, sympathetic outflow from cerebral centers, normal endocrine activity and the presence of an angiospastic humoral substance.

Since Kraus suggested to Brünning [79] the surgical approach to the problem of hypertension, investigations have been made by Daniélopolu,[80] Pieri,[81] Adson and Allen,[82] Craig,[83] Peet,[84] Leriche,[76] Crile,[85] Dandy [86] and Smithwick [87] to find a method of surgical treatment of this disease through extensive sympathectomy. Four types of sympathectomy are in common use today: subdiaphragmatic sympathectomy (Adson and Allen, Craig and Leriche), supradiaphragmatic splanchnicectomy and lower dorsal sympathetic ganglionectomy (Peet), celiac ganglionectomy (Crile) and thoracolumbar sympathectomy (Smithwick, Dandy and

79. Kraus and Brünning, cited by White and Smithwick.[40]

80. Daniélopolu, D.: Chirurgie du système végétatif, Bull. méd. **37**:988, 1923.

81. Pieri, G.: La resezione dei nervi splancnici: Contributo tecnico alla chirurgia del sistema nervoso vegetativo, Ann. ital. di chir. **6**:678, 1927; Tentativi di cura chirurgica dell'ipertensione arteriosa essenziale, Riforma med. **48**:1173, 1932.

82. Adson, A. W., and Allen, E. V.: Essential Hypertension: General Considerations and Report of Results of Treatment by Extensive Resection of Sympathetic Nerves and Partial Resection of Both Suprarenal Glands, in Collected Papers of the Mayo Clinic, Philadelphia, W. B. Saunders Company, 1936, vol. 28, p. 1001. Allen, E. V., and Adson, A. W.: Physiologic Effects of Extensive Sympathectomy for Essential Hypertension: Further Observations, Ann. Int. Med. **11**:2151, 1938.

83. Craig, W. M.: Surgical Approach to and Resection of the Splanchnic Nerves for Relief of Hypertension and Abdominal Pain, West. J. Surg. **42**:146, 1934; Hypertension: A Consideration of Its Surgical Treatment, Brit. M. J. **2**:1215, 1939. Craig, W. M., and Adson, A. W.: Hypertension and Subdiaphragmatic Sympathetic Denervation, S. Clin. North America **19**:969, 1939.

84. Peet, M. M.: The Surgical Treatment of Hypertension, J. internat. de chir. **5**:1, 1940. Peet, M. M.; Woods, W. W., and Braden, S.: The Surgical Treatment of Hypertension: Results in 350 Consecutive Cases Treated by Bilateral Supradiaphragmatic Splanchnicectomy and Lower Dorsal Sympathetic Ganglionectomy, J. A. M. A. **115**:1875 (Nov. 30) 1940.

85. Crile, G.: The Surgical Treatment of Hypertension, edited by A. Rowland, Philadelphia, W. B. Saunders Company, 1938, p. 237; Operative Treatment of Essential Hypertension, South. Surgeon **7**:220, 1938; Progress Notes on the Treatment of Essential Hypertension by Celiac Ganglionectomy, S. Clin. North America **19**:1205, 1939.

86. Bordley, J., III; Galdston, M., and Dandy, W. E.: The Treatment of Essential Hypertension by Sympathectomy, Bull. Johns Hopkins Hosp. **72**:127, 1943.

87. Smithwick, R. R.: A Technique for Splanchnic Resection for Hypertension, Surgery **7**:1, 1940.

Poppen [88]). All these operations have the purpose of interrupting more or less extensively the sympathetic pathways between the thoracolumbar sympathetic chain and the celiac and mesenteric plexuses. Analysis of the results thus far obtained in the surgical treatment of hypertension shows that in certain patients the blood pressure can be lowered in a significant manner by splanchnicectomy. White and Smithwick [40] pointed out that at the present time great emphasis should be placed on the selection of cases for sympathectomy. All factors that may be of value in prediction of a favorable effect from operation must be considered. They emphasized the point that the best index is the reduction in blood pressure.

As the sympathetic operation in the treatment of hypertension causes lowering in blood pressure by interruption of the sympathetic pathways, it is logical to determine preoperatively the effect that follows the temporary interruption of the same sympathetic nerves. On this basis I have tried and I use in cases of hypertension the anesthetic block of the splanchnic nerves and first lumbar ganglion as a preoperative test before performing splanchnicectomy. The anesthetic block is first performed on the left side, the next day on the right side and on the third day bilaterally. The curves of the arterial blood pressure are studied during and between the anesthetic blocks. It was observed that anesthetic block is followed by an immediate effect on the blood pressure, lasting about one hour. The effect obtained is equivalent to that which follows immediately the surgical interruption of the splanchnic nerves and first lumbar ganglion at the same level. This test gives useful indication in each case of hypertension as to the probable result of splanchnicectomy.

It must be remembered that anesthetic block gives information regarding only the immediate vasomotor effect that follows interruption of the splanchnic nerves and the first lumbar ganglion. In experiments carried out on dogs [89] to determine the influence of anesthetic block and of sympathectomy of the lumbar sympathetic chain on the development of the collateral circulation of the lower extremities, we have observed that in the first hours after interruption of the sympathetic pathways the effect on the development of the collateral arterial circulation is similar. However, the study of other dogs weeks or months after lumbar sympathectomy showed that the development of the arterial collateral circulation in the posterior extremities was greater than that in the animals observed only a few hours after interruption of the

88. Poppen, J.: Personal communication to the author.

89. de Sousa Pereira, A.; Rodrigues, A., and Carvalho, R.: La sympathectomie lombaire, l'infiltration novocaïnique lombaire, l'injection intra-artérielle de novocaïne, la résection unilatérale des nerfs splanchniques et la valeur comparée de l'effect de ces interventions sur le dévelopement de la circulation collatérale des membres, Presse méd. **47**:1545, 1939.

sympathetic chain. These experiments suggest that anesthetic block gives an immediate result comparable with the one observed immediately after the same sympathetic nerves are surgically interrupted. Later, however, sympathectomy is followed by a greater development of the arterial collateral circulation. This problem is under study, and the results observed will be presented.

7. In the Postoperative Period of Abdominal Operations: After abdominal operations there is some paresis of the intestine, so that gas does not pass along the bowel to be expelled properly, which may cause distention, pain and at times vomiting (Davis and Hansen [90]). On the other hand, the visceral operative wound during the postoperative period may cause pain produced by the stimulation of the sensory innervation. One of the consequences of visceral pain in the postoperative period is the diminution of the motility of the diaphragm, reducing pulmonary ventilation and favoring pulmonary complications. Complications occur in a much higher percentage after operations on the upper part of the abdomen.[91] In 90 per cent of cases, postoperative pulmonary complications are dependent on the general preoperative condition of the patient and are caused by the excessive formation of bronchial secretion and the retention of this secretion in the bronchi as a result of the operation (King [92]). It was emphasized by Keith [93] that the diaphragm plays an important part in facilitating ventilation of the lower pulmonary lobes, and it was shown by Head,[94] Powers,[95] Carlson [96] and Beecher [97] that pulmonary hypoventilation, with reduction of vital capacity and decreased diaphragmatic excursions, occurs commonly after abdominal operations. Restriction of respiration is an important factor in the cause of postoperative pneumonia (Sise, Mason and Bogan [98]), and the

90. Davis, H. H., and Hansen, T. M.: Investigation of the Cause and Prevention of Gas Pains Following Abdominal Operations, Surgery **17**:492, 1945.

91. Churchill, E. D.: Pulmonary Atelectasis with Special Reference to Massive Collapse of the Lung, Arch. Surg. **11**:489 (Oct.) 1925.

92. King, D. S., in Mason, R. L.: Preoperative and Postoperative Treatment, Philadelphia, W. B. Saunders Company, 1937, p. 192.

93. Keith, A.: The Mechanism of Respiration in Man, in Hill, L.: Further Advances in Physiology, New York, Longmans, Green & Co., 1909, p. 182.

94. Head, J. R.: The Effect of Operation Upon the Vital Capacity, Boston M. & S. J. **197**:83, 1927.

95. Powers, J. H.: Vital Capacity: Its Significance in Relation to Postoperative Pulmonary Complications, Arch. Surg. **17**:304 (Aug.) 1928.

96. Carlson, H. A.: Changes in Respirations Produced by Surgical Operations, Proc. Soc. Exper. Biol. & Med. **29**:23, 1931.

97. Beecher, H. K.: The Measured Effect of Laparotomy on the Respiration, J. Clin. Investigation **12**:651, 1933.

98. Sise, L. F.; Mason, R. L., and Bogan, I. K.: Prophylaxis of Postoperative Pneumonia: Preliminary Report of Some Experiments After Upper Abdominal Operations, Anesth. & Analg. **7**:187, 1928.

varying degrees of partial atelectasis which occur after operations on the upper part of the abdomen are due primarily to diaphragmatic elevation and restriction (Muller, Overholt and Pendergrass [99]). The restriction of pulmonary ventilation appears to be caused by the operative wound which directly and reflexly interferes with abdominal and diaphragmatic excursions (Gius [100]).

Postoperative atelectasis is more frequent and is better explained on a mechanical than on an infectious basis (Blalock [101]), and it is believed that it results from the combined action of weakened respiratory force and bronchial obstruction (Churchill [102]). According to Elliot and Dingley,[103] obstruction of the bronchioles and the reflex immobility of the diaphragm play an important part in the causation of atelectasis. In the postoperative bronchopulmonary infectious complications, the bronchogenous mechanism acts either through the irritating effect of anesthetics administered by inhalation or by stagnation of its contents in the airways. This stagnation results from the inhibitory action of the pain, the forced immobilization and the diminished pulmonary ventilation. The retention of secretions in the tracheobronchial tree is caused by several factors. Inability to cough effectively appears as chief among these (Blalock [101]). Pain at the level of the operative wound in operations on the upper part of the abdomen is a cause of the inability of the patients to cough. All the facts mentioned point out the important influence of pain in the mechanism of atelectasis and other postoperatime pulmonary complications.

Attempts have already been made by some authors to find the most adequate method for reduction or elimination of postoperative pain by physiologic interruption of the pathways of painful sensations between the area of the operative wound and the spinal cord. Crile [104] advised the injection of anesthetic solution (quinine and urea hydrochloride) around the operative wound, which produces analgesia for several days to a week. This method, however, was discarded because it produced fibrinous exudates, with an occasional tendency to suppuration at the

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99. Muller, G. P.; Overholt, R. H., and Pendergrass, E. P.: Postoperative Pulmonary Hypoventilation, Arch. Surg. **19**:1322 (Dec.) 1929.

100. Gius, J. A.: Paravertebral Procaine Block in the Treatment of Postoperative Atelectasis, Surgery **8**:832, 1940.

101. Blalock, A.: Principles of Surgical Care: Shock and Other Problems, St. Louis, C. V. Mosby Company, 1940.

102. Churchill, E. D., and McNeil, D.: The Reduction in Vital Capacity Following Operation, Surg., Gynec. & Obst. **44**:483, 1927.

103. Elliot, T. R., and Dingley, L. A.: Massive Collapse of the Lungs Following Abdominal Operations, Lancet **1**:1305, 1914.

104. Crile, W. A., cited by Allen, C. W.: Local and Regional Anesthesia, ed. 2, Philadelphia, W. B. Saunders Company, 1918, p. 205.

sites of injection. Later Capelle [105] recommended in surgical wounds the continuous administration of procaine hydrochloride to relieve pain and to increase the postoperative vital capacity. In 2 cases of atelectasis following appendectomy in children, Gius [100] obtained remarkable therapeutic results with paravertebral anesthetic block of the operative wound. This procedure allows active hyperventilation of the lungs and effective coughing. The 2 patients were successfully treated by this method. In a series of 8 patients who had had operations on the biliary tract, Starr and Gilman [106] performed anesthetic blocks of the sixth to tenth intercostal nerves according to the technic described by Bartlett.[107] Following the intercostal nerve block, anesthesia persisted for three or four hours and analgesia for more than twenty-four hours. When abdominal pain returned, it was mild in every case. In these patients postoperative pain in wounds in the upper part of the abdomen was relieved, coughing was easier and more effective and there was an increase in pulmonary ventilation. In 15 patients Zollinger [108] practiced anesthetic block, according to the technic of Bartlett,[107] of the sixth to eleventh intercostal nerves, with eucupine solution in oil to obtain anesthesia of the operative wound of the abdominal wall. In 20 cases of operations on the upper part of the abdomen, Belinkoff [109] tried postoperative block of the last intercostal nerves, with Novest-Oil (Monocaine [2-isobutylaminoethyl-*p*-aminobenzoate], benzyl alcohol, ethyl aminobenzoate and expressed oil of almond) as the anesthetic solution. He concluded that this method relieves the postoperative pain and favors pulmonary ventilation, reducing consequently the incidence of postoperative complications.

The role of pain in wounds of the abdominal wall in the mechanism of pulmonary hypoventilation and in the reduction of the diaphragmatic excursion has been already demonstrated (Churchill and McNeil [102]). However, in operations on the upper part of the abdomen, in addition to the parietal pain there is also a deep visceral pain at the level of the visceral wound. It has already been seen that the splanchnic nerves carry the afferent sensory pathways through which pass the painful sensations of the organs innervated by the solar plexus. During

105. Capelle, W.: Die Bedeutung des Wundschmerzes und seiner Ausschaltung für den Ablauf der Atmung bei Laparotomierten, Deutsche Ztschr. f. Chir. **246**: 466, 1936.

106. Starr, A., and Gilman, S.: The Effect of Postoperative Intercostal Nerve Block on Pulmonary Ventilation, New England J. Med. **227**:102, 1942.

107. Bartlett, R. W.: Bilateral Intercostal Nerve Block for Upper Abdominal Surgery, Surg., Gynec. & Obst. **71**:194, 1940.

108. Zollinger, R.: Observations on the Use of Prolonged Anesthetic Agents in Upper Abdominal Incisions, Surgery **10**:27, 1941.

109. Belinkoff, S.: Intercostal Block with Long-Acting Anesthetic in Upper Abdominal Operations, Anesthesiology **5**:500, 1944.

splanchnicectomy performed with the patient under spinal anesthesia, Leriche [45] observed that mechanical stimulation of the upper end of the splanchnic nerves orginated thoracic pain. I have verified the same fact in several patients in whom splanchnicectomy was performed while they were under local anesthesia. Repeated mechanical stimulations of the splanchnic nerves were followed by pain referred to the level of the thorax. This pain was completely absent when the same stimulations were repeated after anesthetic block of the splanchnic nerves. On the other hand, I have also demonstrated that in certain cases of angina pectoris precordial pain may be originated in stimulations arising at the level of the abdominal organs innervated by the solar plexus. The precordial pain was temporarily relieved by anesthetic block of the splanchnic nerves and definitively by splanchnicectomy.

The facts mentioned previously led me to try repeated anesthetic blocks of the splanchnic nerves to relieve during the postoperative period the visceral pain carried through the afferent sensory pathways of the splanchnic nerves. Two years ago I began studying the role of the sympathetic innervation in the postoperative period of abdominal operations in its relation to visceral pain and postoperative reflexes originating in the visceral wound. These investigations were carried on especially in operations on the upper part of the abdomen, performed in organs innervated by sympathetic branches of the solar plexus. Repeated anesthetic blocks of the splanchnic nerves and first lumbar ganglion were performed during the four or five days that followed the abdominal operation. Normally the first anesthetic block was performed twelve to twenty-four hours following the operation. The next blocks were performed every day on the left side, on the right side or bilaterally, according to the topography of the sympathetic innervation in relation to the topography of the visceral operative wound. These anesthetic blocks were repeated until the patient could breathe easily, without deep abdominal pain.

In 35 patients submitted to different abdominal operations (gastrectomy, gastroenterostomy, cholecystectomy, colectomy and appendectomy), this postoperative treatment of pain was routinely carried out. It was observed in these cases that the deep visceral pain was immediately and completely, although temporarily, relieved in every case after unilateral or bilateral anesthetic block of the splanchnic nerves, according to the topography of the visceral operative wound. During the relief of pain the patient could breathe deeply and cough more easily. After the first anesthetic block, pain reappeared but was not so intense as before the treatment. After repeated anesthetic blocks, the deep visceral pain was quickly relieved. The motility of the diaphgram following the anesthetic block of the splanchnic nerves showed an increase when the interruption of the splanchnic nerves and the first lumbar ganglion was

performed (fig. 19). In this series of abdominal operations (stomach, biliary tract, intestines and appendix), postoperative pulmonary complications or thrombophlebitis were not observed. The patients were turned frequently and encouraged to breathe deeply, and early ambulation, on the third or fourth day, was permitted for the greater number of these patients.

Another problem is being studied in connection with the relief of postoperative visceral pain and visceral reflexes. The problem is to know whether repeated anesthetic blocks of the sympathetic innervation performed during the postoperative period of the abdominal operations can be useful to prevent or to lessen the occurrence of postoperative phlebitis. It is admitted at the present time that interruption of the sympathetic innervation of the veins by repeated anesthetic blocks is a useful method in the treatment of postoperative phlebitis. Interruption of the sympathetic innervation of the veins in phlebitis and throm-

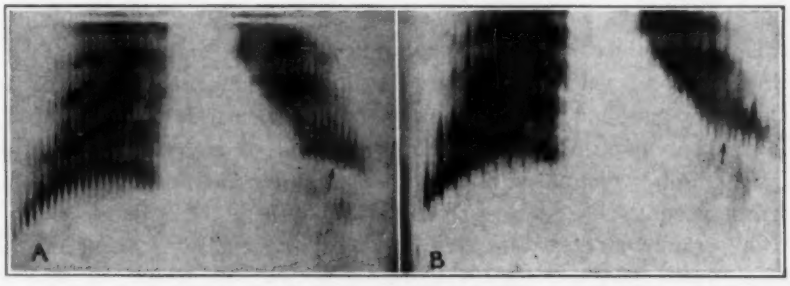

Fig. 19.—Ulcer of the lesser curvature in a patient on whom gastrectomy was performed. *A*, kymography of the diaphragm forty-four hours after gastrectomy. *B*, kymography on the same patient twenty minutes after the anesthetic block of the left splanchnic nerves. Note the increased amplitude of the left hemidiaphragm after the splanchnic block.

bophlebitis of the lower extremities is followed by relief from pain, relief from venospasm and increase in the collateral venous circulation.[110]

In 15 patients submitted to hysterectomy, the nature of the operation and the condition of the patients led to the supposition that phlebitis might occur as a postoperative complication. Anesthestic blocks of the splanchnic nerves and first lumbar sympathetic ganglion were repeated during one to two weeks, according to the postoperative progress of each patient. This treatment was associated with an early ambulation of these patients. In none of the cases did phlebitis occur. The results obtained in these few cases do not yet permit a conclusion; the problem continues under investigation.

110. de Sousa Pereira, A.: Innervation of the Veins: Its Role in Pain, Venospasm, and Collateral Venous Circulation, to be published.

SUMMARY

Anesthetic block of the splanchnic nerves or of these nerves and the first lumbar ganglion has aroused in recent years a great interest in the study and solution of clinical problems related to these nerves.

Of the two routes of approach available today for the blocking of the splanchnic nerves, the posterior approach was practiced routinely in these investigations. When repeated anesthetic blocks were indicated, the needle was inserted not always at the same point, as advised in previous technics of other authors, but at different points in an area determined according to the topography of the splanchnic nerves and the first lumbar ganglion in relation to the skeleton.

The technic followed in the block of the splanchnic nerves and first lumbar ganglion is described, and the accidents observed with this anesthetic block are reported.

Although the clinical indications of single or repeated anesthetic blocks of the splanchnic nerves are still under investigation, encouraging results were obtained in the diagnosis and treatment of patients with cardiospasm, visceral pain and peptic ulcer.

Single or repeated anesthetic blocks of the splanchnic nerves and first lumbar ganglion were tried in cases of ileus paralyticus, megacolon, hypertension and in the postoperative period of abdominal operations as as method of diagnosis, as a method of treatment or as a test before splanchnicectomy.

The results already observed in these investigations point out the clinical importance of anesthetic block of the splanchnic nerves and first lumbar ganglion.

PULMONARY SUCK AND BLOW AS A RESPIRATORY ANALEPTIC

Interdependence of Cardiac Massage and Suck and Blow Resuscitation

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IN considering the problem of resuscitation, one is prone to regard cardiorespiratory stimulants as chemical. The fact that certain analeptics may be purely mechanical or reflex in their *modus operandi* has received little or no clinical consideration. It is the purpose of this paper to present the results of studies supplementing a series of studies previously made by Thompson and Birnbaum [1] on a mechanical method of respiratory stimulation.

Mechanical devices have been used in pulmonary resuscitation since the middle of the sixteenth century, the time of Paracelsus,[2] who

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Read May 16, 1945, before the Surgical Division, Clinical Research Meeting, New York Academy of Medicine. The presentation was made by Dr. Joseph H. Fobes, director of the Graduate School of Surgery, New York Medical College, Flower and Fifth Avenue Hospitals.

Experimental studies were performed with the collaboration of Dr. Joseph A. Abbey, who is now a captain in the Medical Corps, Army of the United States.

Dr. S. A. Thompson gave invaluable suggestions during the course of these investigations.

1. (*a*) Birnbaum, G. L., and Thompson, S. A.: The Mechanism of Asphyxial Resuscitation, Surg., Gynec. & Obst. **75**:79 (July) 1942, (*b*) Thompson, S. A., and Birnbaum, G. L.: Phenomenon of Asphyxial Resuscitation: Resuscitation with Inert (Asphyxiating) Gases, Proc. Soc. Exper. Biol. & Med. **48**:203 Oct. 1941; (*c*) Resuscitation in Advanced Asphyxia, J. A. M. A. **118**:1364 (April 18) 1942; (*d*) The Phenomenon of Asphyxial Resuscitation, Surg., Gynec. & Obst. **74**:1078 (June) 1942; (*e*) Resuscitation in Advanced Asphyxia, Surgery **12**:284 (Aug.) 1942; (*f*) Asphyxial Resuscitation: The Phenomenon and Its Mechanism, J. Thoracic Surg. **12**:624 (Oct.) 1943; Comparative Value of Various Methods of Resuscitation, Ann. Surg. **120**:94 (July) 1944.

2. (*a*) E. and J. Resuscitator and Inhalator Acceptable, report of the Council on Physical Therapy, J. A. M. A. **112**:1945 (May 13) 1939. (*b*) Harrison, W.: A Brief Historical Review of the Employment of a Bellows as a Means of Inducing Artificial Respiration During the Three Hundred Years Which Elapsed Between A. D. 1530 and 1830, Detroit M. J. **16**:341 (Aug.) 1916.

utilized a fireside bellows. Present appliances fall into two main groups: (1) those of the Drinker type, applied externally to the thoracic wall, and (2) those of the pulmotor variety, which are utilized directly on the tracheobronchial tree. The latter, in turn, may be subdivided into appliances which inflate the lungs and those which utilize suction and/or inflation.

It is beyond the scope of this paper to enter into a detailed discussion of the pros and cons of the various mechanical methods. Most of the controversy centers on the desirability of active pulmonary deflation as opposed to that of dependence on passive elastic recoil of the lungs and thoracic wall for the accomplishment of exhalation. Thompson and Birnbaum, in their work as protagonists of suction in pulmonary resuscitation, have recently shown that alternating suction-inflation causes a reflex stimulus to respiration. To demonstrate this fact they utilized an inert asphyxiant gas—nitrogen or helium—in order to avoid any possible respiratory stimulation by the gas itself. This has been described by them as the "phenomenon of asphyxial resuscitation." Expressed differently, one might say that they demonstrated the existence of a mechanical or reflex respiratory analeptic.

That the application of suction alone to the trachea and bronchi is a potent respiratory stimulant is shown in figure 2. It will be seen that each of a series of suction strokes causes an inspiratory gasp, irrespective of the phase of respiration. No such effect takes place with a similar series of pressure strokes. In other words, suction is utilized as a stimulus, not, as Henderson expressed the opinion, as a means of aspirating gas from the lungs.[3]

External methods of artifical respiration (manual and mechanical) do not stimulate; they merely duplicate respiratory movements until the normal physiologic nervous and chemical elements in control are able to take effect. Mechanical devices applied externally serve only as substitutes for prolonged manual artificial respiration and, as such, are not stimulatory. It should be repeated and reemphasized that the mechanical devices which act directly on the pulmonary tree, particularly in the application of suction, are stimulatory and not substitutive.

Concerning cardiac resuscitation, it can probably be said with assurance that this may most effectively be accomplished by direct manual stimulation of the heart. Both Coryllos [4] and Birnbaum and Thompson [1a] have demonstrated the importance of maintaining pulmonary ventilation during massage. However, several of my experiments (fig. 1) indicate that such a combination would frequently be inadequate if either pressure

3. Henderson, Y., and Turner, J. M.: Artificial Respiration and Inhalation, J. A. M. A. **116**:1508 (April 5) 1941.

4. Coryllos, P. N.: Mechanical Resuscitation in Advanced Forms of Asphyxia, Surg., Gynec. & Obst. **66**:698 (April) 1938.

alone or suction alone were applied to the respiratory passages. Both suction and inflation should be utilized with cardiac massage.

PHYSIOLOGY OF RESPIRATION

Control of respiration is both reflex and chemical, and both mechanisms exert their influence on the respiratory center. Nervous control is mediated chiefly through the vagi, the influence of which is inhibitory; section of these nerves abolishes their inhibitory control. Both inflation and deflation reflexes have been demonstrated. However, under ordinary conditions only the inflation reflex is functional. During suction deflation a new set of end organs comes into play; these are more effective in resuscitation than are the stretch receptors of inflation.[1a, f] Galvanometric studies on the distal cut end of the vagus demonstrate the presence of action currents on inflation and suction but none on mere passive expiration.[5] Resuscitation by suck and blow fails to occur after the vagi are sectioned.

Chemical control is exerted chiefly by the relative concentrations of carbon dioxide and oxygen. Pulmonary ventilation is increased by carbon dioxide stimulation of the respiratory center. However, the center is said to be depressed by carbon dioxide in the presence of asphyxia.[1a]

ASPHYXIA

A complete discussion on the mechanism of asphyxia will not be entered on. Lougheed, Janes and Hall [6] found that in asphyxiated dogs the critical interval after the cessation of respiration was eleven to seventeen seconds.

Thompson and Birnbaum [1f] stated that the critical point for spontaneous recovery falls within thirty to forty-five seconds; in most cases an extension of only a few seconds rendered recovery impossible, even with manual artificial respiration combined with oxygen–carbon dioxide mixtures. On the other hand, a delay of one half to two and three-fourths minutes is possible, with recovery, if suck and blow is substituted for manual methods.

Four phases of asphyxia are mentioned by Coryllos.[4] These phases are, successively: (1) initial apnea, in which blood pressure rises and the breath is held, with or without spasm of the glottis; (2) dyspnea, characterized by labored, increased respiratory movements, with utilization of the accessory muscles of respiration, continued rise of blood pressure and perhaps tonic and clonic convulsions; (3) terminal apnea, with respiratory cessation, drop in blood presure, loss

5. Birnbaum and Thompson.[1a] Thompson and Birnbaum.[1e, f]

6. Lougheed, D. W.; Janes, J. M., and Hall, G. E.: Physiological Studies in Experimental Asphyxia and Drowning, Canad. M. A. J. **40**:423 (May) 1939.

of muscle tone, pupillary dilatation and areflexia, with loss of sphincter control, and (4) cardiac failure, characterized by either ventricular standstill or fibrillation.

Manual artificial respiration at the end of phase 3 is ineffective, but suck and blow at this time causes a rise in blood pressure within thirty seconds. If pulmonary ventilation is maintained after cardiac arrest, then cardiac massage is effective even ten minutes later.

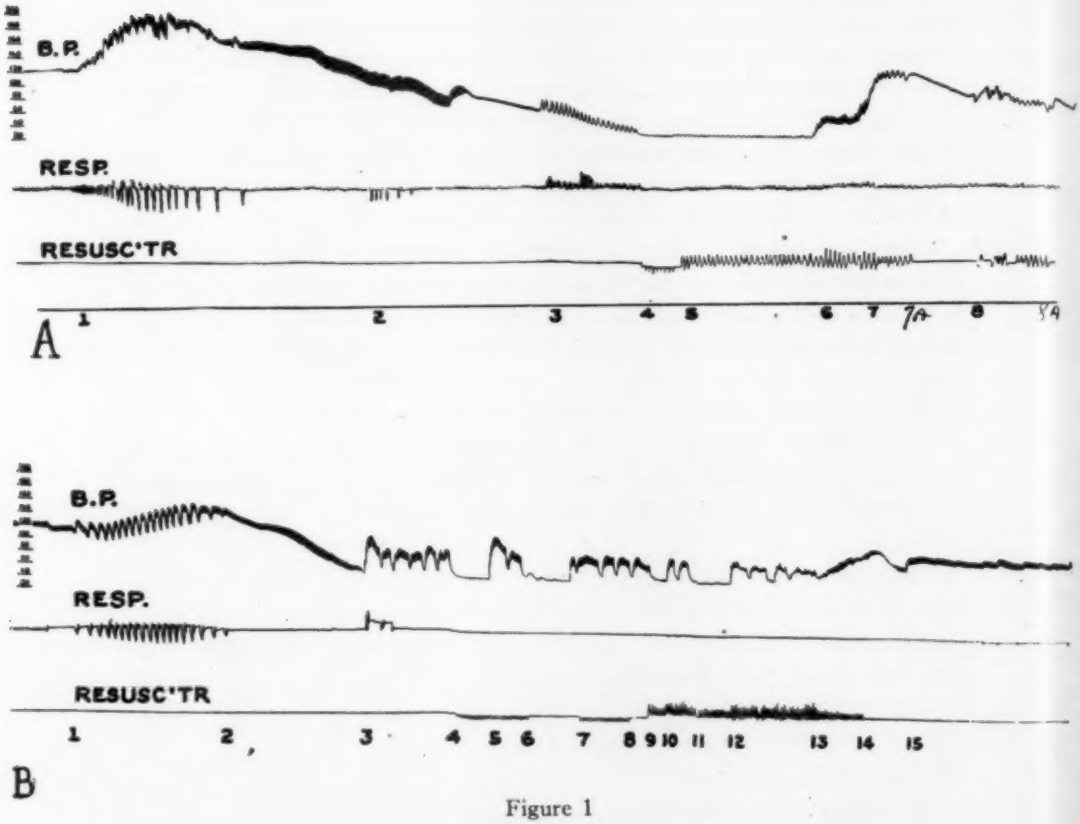

Figure 1

(See legend on opposite page)

HISTORY OF SUCK AND BLOW DEVICES

John Hunter [7] first used a double-chambered bellows experimentally as a suction-inflation resuscitator in 1755, and he recommended its use clinically in 1776. The use of a single-chambered fireside bellows for insufflation was popular in Europe and had been mentioned by Paracel-

7. Hunter, J.: Works, edited by J. F. Palmer, London, Longman [and others], 1837, vol. 3.

sus in 1560.[2] Courtois[8] in 1790 devised a hand-operated suck and blow pump, which he called *pompe apodopnique,* literally, a resuscitation pump. This is the earliest forerunner of the lungventilator, used in my experiments.

EXPLANATION OF PLATE.

Fig. 1.—*A,* The experiment demonstrates successful cardiac resuscitation by means of transdiaphragmatic massage together with inflation and deflation of the lungs. Air was used in the series of experiments presented in this paper. Resuscitation was accomplished after several other methods had failed. The top line represents the blood pressure, the middle line the respiration and the third the lung ventilator. At 1 obstructive asphyxia was produced by clamping the endotracheal tube. At 2 a few terminal gasps occurred and then respirations ceased. At 3 manual artificial respiration was attempted, without success, and the blood pressure continued to fall. At 4 suction and release were instituted by use of the suction tube of the apparatus and were continued to 5, without success. During these periods there did not appear to be any heart beat. At 5 positive inflation and negative deflation of the lung were instituted up to 6, without success.

At 6 a high midline incision was made in the abdomen and two fingers were slipped under the diaphragm; transdiaphragmatic massage was instituted at the same time that inflation and deflation of the lungs were continued. Notice the almost immediate resuscitation of the cardiovascular system; the elevation of the blood pressure was maintained. At 7 cardiac massage was discontinued. At 7A the use of the lungventilator was discontinued. Notice the downward trend of the blood pressure. At 8 a few more strokes of the lungventilator were instituted, and then they were discontinued at 8A, as recovery appeared to be maintained.

This experiment shows the necessity of cardiovascular resuscitation as well as respiratory resuscitation in the late stages of asphyxia. Three immediately preceding methods of respiratory resuscitation had failed completely to resuscitate the animal, although they did produce pulmonary ventilation. Without this pulmonary ventilation, cardiac massage would not have been successful. *B,* this experiment again demonstrates the necessity of cardiovascular as well as respiratory resuscitation in the late stages of asphyxia. Figure 1 demonstrates the failure of respiratory resuscitation alone and cardiac resuscitation alone and their success when combined. It also shows the necessity for pulmonary suck and blow ventilation in successful cardiac resuscitation. The technic is similar to that described in *A.* The top line represents the blood pressure, the middle line represents the respiration and the bottom line represents the lungventilator. At 1 tracheal obstruction is produced by clamping the endotracheal tube. At 2 respirations have ceased. At 3, through a median abdominal incision, transdiaphragmatic cardiac massage is attempted, no pulmonary ventilation being used, and is unsuccessful. At 4 suction and release alone is used and is unsuccessful. At 5 suction is combined with transdiaphragmatic cardiac massage and is unsuccessful. Suction is discontinued at 6; suction and transdiaphragmatic cardiac massage are again continued, from 7 to 8, but are unsuccessful. At 9 inflation and release together with transdiaphragmatic cardiac massage is attempted but is unsuccessful. At 11 transdiaphragmatic cardiac massage is discontinued. At 12 transdiaphragmatic cardiac massage together with positive inflation and negative deflation of the lung is started. This is continued to 13, where successful cardiovascular resuscitation occurs, as shown by the elevated and maintained blood pressure.

In many of these experiments, deliberate attempts were made to damage the dog's lungs by the exertion repeatedly of either maximally forceful downstrokes and/or upstrokes (pressure and suction respectively). Careful examination of the entire pulmonary tree failed to disclose any injury.

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8. Courtois, M. L. H.: Mémoire sur les asphyxies (a), avec la description d'un nouvel instrument propre à rappeler le méchanisme de la respiration, J. de méd., chir., pharm. **82**:361, 1790.

The first practical automatic suck and blow device energized by compressed gas was the pulmotor, which appeared in 1911. Booher [9] in 1915 demonstrated a hand-operated apparatus, called lungmotor, consisting essentially of two cylindric pumps. However, no active suction was effected by this apparatus. The E. and J.,[10] Emerson [11] and McKesson [12] resuscitators are gas-activated suck and blow devices of the pulmotor type.

The suck and blow apparatus utilized in my experiments is called the lungventilator. This breathing machine is operated as a hand pump.

It consists of two separate pumps, each barrel and piston unconnected with the other. One pump, or one side of the machine, is used for inflation and creates a maximum pressure of 20 mm. of mercury. The other side of the machine is used for suction deflation and creates a maximum suction of minus 10 mm. of mercury. The maximum pressure is controlled by a blow-off ball valve which vents at 20 mm. of mercury. The suction likewise is controlled as to its maximum by a value that vents at 10 mm. This prohibits the building up of pressures greater than those quoted. That these pressures are safe is borne out by the fact that in this series of one hundred and forty-three experiments on 40 dogs no evidence of injury to the lungs was found at the autopsy of any of the animals. The safety of these pressures has repeatedly been corroborated.[13] The pressure and suction created by the lungventilator is conveyed, through semirigid tuning, to a Y tube immediately outside of a face mask and then into the mask itself. There is an additional safety blow-off valve in the face mask, which operates at a pressure of plus 40 mm. of mercury. Since the pressure and suction tubes are connected separately to the Y tube, either one of the tubes can be disconnected as desired, and, by alternately closing and opening the disconnected end of the Y tube, the machine can be converted and used for inflation and release or suction and release of the lung. This is a desirable added advantage.

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9. Booher, J. W.: Demonstration of the Lung-Motor, Dent. Summary **36**: 948, 1916.

10. E. and J. Resuscitator, Inhalator, Aspirator (Fox Model) Acceptable, report of the Council on Physical Therapy, J. A. M. A. **121**:1219 (April 10) 1943; Footnote 2*a*.

11. Emerson Resuscitator Hospital and Portable Models Acceptable, report of the Council on Physical Therapy, J. A. M. A. **119**:414 (May 30) 1942.

12. Steinberg, B., and Deitz, A.: Manual and Mechanical Resuscitation in Experimental Asphyxia, J. Lab. & Clin. Med. **29**:695 (July) 1944.

13. Final Report of the Commission on Resuscitation from Carbon Monoxide Asphyxia: The Treatment of Carbon Monoxide Asphyxia, J. Indust. Hyg. **5**:125 (Aug.) 1923. Henderson, Y.: Resuscitation Apparatus, J. A. M. A. **67**:1 (July 1) 1916. Ross, B. D.: A Survey of Methods for Artificial Respiration, ibid. **122**: 660 (July 3) 1943. Henderson and Turner.[3] Coryllos.[4]

Two separate lungventilator resuscitators were examined and used in these experiments. The two machines are essentially similar in their construction, appearance and operation. The principal difference is that one machine has a total capacity of 600 cc. per stroke and the other machine has a total capacity of 1,000 cc. per stroke. The maximum pressure and suction are the same in the two machines. However, in the machine of 600 cc. capacity, by the removal of the steel balls in the ball valve sockets and the substitution of aluminum balls the maximum pressure is reduced from 20 to plus 10 or 12 mm. of mercury and the maximum suction is reduced from minus 10 to minus 4 to 6 mm. of mercury. This of itself does not appear to have any advantage.

In these experiments the technic was as follows:

Medium-sized dogs were anesthetized with pentobarbital sodium given intraperitoneally (1 cc. per 5 pounds [2.3 Kg.]). The femoral artery was then cannulized and connected to a recording mercury manometer which registered on a smoked drum. A small metal intrapleural cannula was then inserted into the pleural space, under air-tight conditions. This cannula was connected directly to a recording mercury manometer which registered the respirations as well as the intrapleural pressure. A close-fitting endotracheal tube with an occlusion cuff was then inserted into the trachea of the animal. After being properly placed, the occlusion cuff was then inflated, thus making the endotracheal tube completely leakproof. The mechanical resuscitator was then connected by means of a T tube to the endotracheal tube and to a recording mercury manometer which registered on the same smoked drum. Asphyxia was produced by two methods: (1) inhalation of 100 per cent nitrogen gas or (2) clamping of the endotracheal tube. The animal was asphyxiated and allowed to go to a "critical point." This "critical point" occurred in the third stage of asphyxia, with a rapidly falling blood pressure and definite changes in the rhythm and output of the heart beat. At this "critical point," various resuscitation procedures were attempted.

EXPERIMENTAL DATA

The chief purpose of this paper is the experimental demonstration of the importance of combining suck and blow with cardiac massage in resuscitation. A series of preliminary experiments were performed for the purpose of studying the lungventilator and to determine the value of suction and suck and blow in resuscitation. These findings differed in no essential from those demonstrated by Thompson and Birnbaum. Suffice it to say that in some experiments deliberate attempts were made to damage the lungs of the animals by exerting maximally powerful pressure and suction strokes. No injury could be demonstrated at autopsy.

COMMENT

The usual operating room practice in the presence of cardiorespiratory failure during a laparotomy or any other major procedure

is to inject chemical analeptics and to administer oxygen or oxygen–carbon dioxide mixtures by direct flow, i. e., positive pressure, with or without manual artificial respiration. If the experimental findings presented may be regarded as applicable to human physiology, then one must conclude that two vitally important elements are ignored in resuscitation. One is cardiac massage, and the other is the use of suction-inflation in ventilating the lungs.

Figure 1 *A* demonstrates that cardiac massage alone, massage with pulmonary suction and massage with pulmonary inflation fail to resuscitate until each is combined with suction-inflation. In figure 1 *A*, preliminary resuscitory measures were entirely pulmonary; these failed until cardiac massage was added to suction-inflation. Figure 2 illustrates

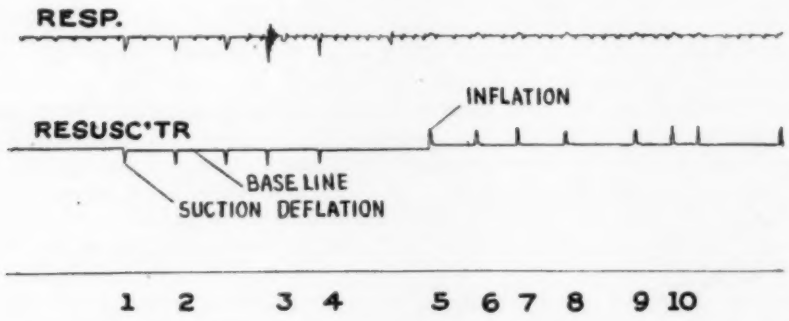

Fig. 2.—The experiment shows the effect of suction and release on the respirations of the breathing anesthetized dog and also the effect of inflation and release on the respiration of the breathing anesthetized dog. When inflation alone is used, there does not seem to be any expiratory or inspiratory stimulation and there is little or no change in the rhythm of the respiration. When suction alone is used, this causes an immediate inspiratory gasp, which is pronounced and is followed by a definite change in the rhythm of respiration.

The technic here is similar to that described in figure 1 *A*. The top line represents the respirations, and the second line represents the lungventilator. Suction was produced by use of the suction tube alone of the lungventilator, and inflation was produced by use of the inflation tube.

At 1, 2, 3 and 4 suction was produced by the apparatus. Notice that after the immediate release of suction there is an inspiratory gasp by the animal, which is decided and pronounced, particularly at 3. Notice also that there is a prolongation after each gasp, before the next inspiration occurs. At 5, 6, 7, 8, 9 and 10 inflation and release was produced. Notice that there was little respiratory effort after the release of inflation other than the normal passive expiration. There was also little or no change in the rhythm of the respiration. The inference here is that suction produces a much stronger respiratory stimulation than does inflation. This again shows the necessity of combining suction with inflation in resuscitation.

that a single suction stroke causes a reflex inspiratory gasp. Pressure strokes cause no such respiratory stimulation. In other words, while pressure alone ventilates, suction alone acts as a respiratory stimulant, i. e., a physical or reflex respiratory analeptic.

CONCLUSIONS

1. While Coryllos and Thompson and Birnbaum have demonstrated the importance of combining cardiac massage with pulmonary ventilation in resuscitation, my experiments indicate that such a combination would frequently be inadequate if the pulmonary measures consisted in either pressure or suction used alone. The clinical implications are that suck and blow is the ventilation of choice when combined with cardiac massage.

2. In the appropriate circumstances, both cardiac and respiratory stimulation should be used in asphyxia. Either alone is often ineffective.

3. Unlike pressure, suction exerts a stimulatory effect on respiration as indicated by the inspiratory gasp caused by a suction stroke. No such effect is noted with manual artificial respiration.

WAR WOUNDS OF ARTERIES

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DURING the course of one year 115 patients with arterial wounds were admitted to a general hospital in a Zone of Communications. The interval between wounding and admission to this hospital was between a few hours and three weeks. All patients had had emergency treatment and many had had definitive treatment by the time they arrived.

The following types of cases are considered in this series: (*a*) cases of injury to the arteries which control the main blood supply to a limb or

One Hundred and Fifteen Injuries to Major Arteries

Artery	Number	Gangrene	Atrophy	Arterial Operation	Gangrene Followed Injury	Aneurysmorrhaphy	Lateral Suture	Venous Graft	Excision Aneurysm	Simple Ligation
Carotid:										
Common	4	0	2	2	0	1	0	0	0	1
Internal	4	0	4	1	0	0	0	0	0	1
External	3	0	0	3	0	0	0	0	0	3
Subclavian	1	0	0	1	0	0	0	0	1	0
Axillary and brachial	51	10	6	11	0	2	0	0	9	0
Femoral	18	12	0	2	1	0	0	0	0	2
Profunda femoris	4	0	0	4	0	0	0	0	1*	3
Popliteal	30	22	4	3	2	0	0	1	0	2
Totals	115	44	16	27	3	3	0	1	11	12

* This operation required a lateral suture of the common femoral artery.

the brain; (*b*) cases of injury to any artery in addition to the aforementioned which resulted in gangrene, ischemic atrophy or ischemic paralysis; and (*c*) cases of secondary arterial hemorrhage which needed surgical intervention, regardless of the artery involved. The arteries of the forearm and lower leg below the bifurcations of the brachial and the popliteal arteries are not considered. Injury to one of them alone does not affect the circulation, and injury to all of them simultaneously is accompanied with such extensive destruction of soft tissue and bone as to amount nearly to a traumatic amputation.

The table gives a composite view of the material considered. The cases of secondary hemorrhage are not included in the table because most of them duplicate those listed in other columns.

The relative incidence of wounds of the different arteries as shown here cannot be accepted as representative of casualties as a whole. The hospital into which these patients were received was, in addition to its other duties, a neurosurgical center. The high incidence of wounds to peripheral arteries, particularly those of the upper extremity, is presumably explained by the fact that many of the patients were referred to the hospital for care of associated injuries to peripheral nerves.

A. OPERATION ON MAJOR ARTERIES

1. Carotid Arteries.—The serious import of the neurologic complications of injuries to the common and internal carotid arteries earns them special consideration. It is of note that the diagnosis of cerebral damage due to ischemia in these cases is occasionally not thought of. The most striking features that these patients present are the neurologic signs of hemiplegia and aphasia. A wound in the neck may be one of many and so may be belittled. And even if it is the only one there is a great temptation to assume at first glance that the injury to the brain is one of concussion or contusion implemented somehow or other through this wound. In 1 of our patients the diagnosis of "contrecoup injury to the brain" had been made and persisted in even though a lumbar puncture had shown "crystal clear" fluid. Makins [1] stated that in the first World War he heard of a craniotomy being performed in such a case.

All four injuries to the internal carotid artery were followed by hemiplegia, which persisted while the patients were in the hospital, a period of between two and three months. At the end of that time all were improving and were able to walk unattended but with a hemiplegic gait. Curiously, while these wounds were to the left internal carotid artery and all the patients were right handed, only 3 of them had aphasia. Encephalography was done on 3 patients including the patient who had no aphasia, and pronounced dilatation of the left ventricle was seen in all (fig. 1). The explanation of the absence of aphasia in the 1 case has not been made. Two of the patients showed aphonia, 1 having paralysis of the right vocal cord and the other paralysis of the left. Both of these patients arrived with tracheotomy tubes in place, made necessary by the obstruction to the airway by the paralyzed cords. In the case of the patient having the left cord paralyzed, the paralysis can, from the course of the wound, be explained only by injury to the right recurrent laryngeal nerve.

It is of interest to speculate on the nature of the arterial injury in these 4 cases. It is our impression that the circulatory deficiency was due to arterial spasm from contusion. All 4 patients presented

1. Makins, G. H.: On Gunshot Injuries to the Blood-Vessels, Bristol, J. Wright & Company, 1919.

small penetrating or perforating wounds of the neck and little or no hematoma. In 1 of them only was there an indication for exploration (severe secondary hemorrhage from the base of the tongue). Here a segment of the internal carotid artery 1 cm. in length and about 1 cm. distal to its origin from the common carotid artery was found to be constricted to about 60 per cent of the caliber of the vessel proximal to it. The artery distal to this constriction was only slightly larger. The area was transmitting blood. The interpretation was that a

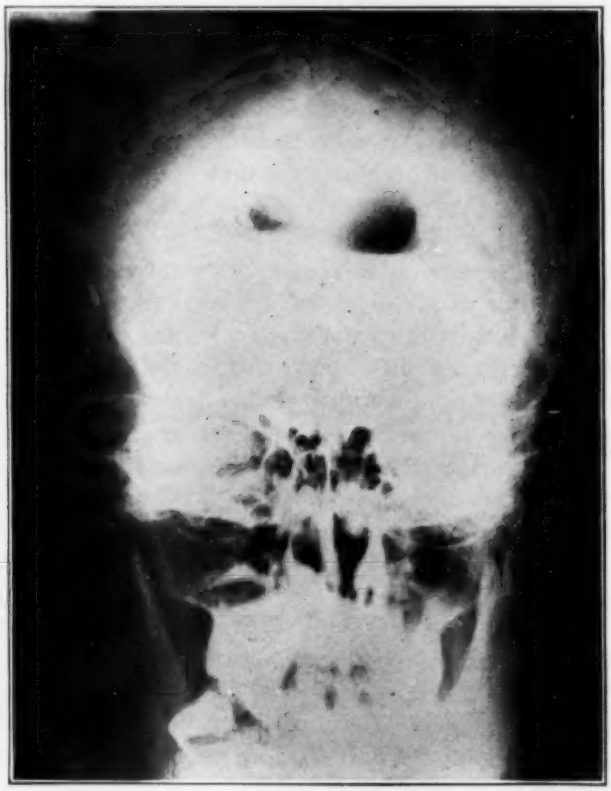

Fig. 1.—Encephalogram of a patient having unilateral internal hydrocephalus from injury to the left internal carotid artery.

contusion of the artery had caused spasm which had persisted and had become involved in scar so that it could not again attain its normal caliber. The initial acute deficiency in blood flow to the brain, possibly in conjunction with sytemic shock, had been enough to cause damage to the left cerebral hemisphere. Similar conditions have been observed in other arteries, severe spasm being described at the initial operation in the forward hospital and found to be still maintained by scar tissue two months later, when exploration was done for other reasons (opera-

tion on the peripheral nerve). The 3 other cases of injury to the internal carotid artery were similar except that in all of them the course of the missile was close to the base of the skull.

Of the 4 patients with injuries to the common carotid artery, 2 suffered transient hemiplegia, which disappeared completely in a few days. In 1 of these the left common carotid artery was injured. The patient suffered aphasia, which disappeared as the hemiplegia decreased. He presented paralysis of the left vocal cord due to injury of the left recurrent laryngeal nerve. The other patient had injury to the right common carotid artery and paralysis of the right cord from damage to the recurrent laryngeal nerve on that side. The other 2 patients had arteriovenous fistulas between the common carotid artery and the internal jugular vein. In them there were no neurologic or other complications.

The nature of the arterial lesion in these injuries to the common carotid artery appeared to be more obvious than in those to the internal carotid artery. Of the 2 patients presenting transient hemiplegia, 1 had a tremendous pulsating hematoma of the neck due to a tear involving over one-half the circumference of the common carotid artery 2.5 cm. below its bifurcation. Ligation of the artery was performed four days after wounding and one day after admission to this hospital. The ligation apparently did not arrest the recovery of the hemiplegia. The other had a traumatic aneurysm, 3 by 5 cm., just above the sternoclavicular joint on the right. A septic pulmonary infarction and empyema developed, and the patient was evacuated, two and one-half months after being wounded, to the United States for further treatment. One of the 2 patients without hemiplegia had a small arteriovenous fistula of the right common carotid artery and the internal jugular vein. Aneurysmorrhaphy was performed. The result was good, and the patient was discharged to duty. The other presented a larger arteriovenous aneurysm, a severe wound with loss of tissue of the face and mandible, and was evacuated for later elective operation.

The testimony of this small group of cases confirms the common teaching that injury to the internal carotid artery is more serious than that to the common carotid artery so far as the resultant damage to cerebral tissue is concerned. The syndrome with hemiplegia, no hematoma in the neck, fracture of the mandible, injury to the vagus nerve or its branches (laryngeal paralysis), injury to the cervical portion of the sympathetic system (Horner's syndrome), clear spinal fluid and normal roentgenograms of the cranial vault suggests injury to the internal carotid artery. An injury to the common carotid artery is perhaps distinguished from this by the commoner occurrence

of pulsating hematoma, Horner's syndrome and injury to the vagus nerve and by the comparative rarity of mandibular fractures and hemiplegia.

II. Subclavian Artery.—Only 1 patient was admitted with injury to the subclavian artery. There was an associated partial paralysis of all cords of the brachial plexus. The arterial lesion was a traumatic aneurysm (6 by 10 cm.) from a wound of the second portion of the artery. This was excised after ligation of the first and third portions of the artery and ligation of the subclavian vein. A neurolysis of the brachial plexus was performed. The clavicle was resected in its middle third to accomplish this. There was no change in the circulation of the hand following operation other than that the radial pulse diminished but did not disappear. A dorsal sympathectomy had been done nine days prior to the attack on the aneurysm. This caused to disappear a considerable degree of causalgia which had previously been present.

The interest in the subclavian artery centers around the technic of an operation which in World War I was supposed to carry a prohibitive mortality. The danger then was recognized to be from hemorrhage and shock, not from gangrene. Our impression is that the ligation of the subclavian artery is technically as easy as that of any other artery provided four things are insisted on: (1) adequate exposure, (2) good anesthesia given intratracheally, (3) blood transfusion and (4) patience to operate for from four to six hours if necessary. During World War I the first point was the only one stressed, chiefly because points 2 and 3, on which point 4 depends, were not available, as they are now. The angular incision of Fiolle and Delmas [2] with resection of the clavicle, particularly on the left side, where the vessels are more inferior, should be routine.

III. Axillary and Brachial Arteries.—There were forty-one injuries to the brachial artery and ten to the axillary artery. The dividing line between these two arteries is the outer border of the teres major,[3] and classically this point is distal to the anterior and posterior circumflex humeral branches. The branches of the axillary and brachial arteries are well known to be subject to such an infinite variety of anomalies, however, that any nominal distinction between the two arteries as to which is the most dangerous to tie is rendered unimportant. Even the consideration as to whether the ligation is done above or below the profunda brachii is of little significance if one compares it with the importance of the intact collateral circulation, frequently a doubtful factor in large traumatic

2. Fiolle, J., and Delmas, J.: The Surgical Exposure of the Deep-Seated Blood-Vessels, London, William Heinemann, Ltd., 1921.

3. Gray, H.: Anatomy of the Human Body, ed. 22, Philadelphia, Lea & Febiger, 1930, p. 587.

wounds of the arm. It is my experience that ligation of the brachial or axillary artery above or below the profunda brachii in a young patient with intact collateral vessels is completely safe.

The frequency of associated injury to the nerve is evident when it is realized that of the 41 patients with injury to axillary or brachial arteries who did not have such extensive gangrene that they could be examined for neural injury, only 1 did not have an accompanying neural lesion. There were 49 patients admitted to the hospital during this period who had injuries to the brachial plexus without vascular injury which could be detected clinically or appeared in the record. About 50 per cent, then, of injuries to the brachial plexus are accompanied by injuries to the axillary or brachial artery, whereas almost 100 per cent of injuries to these arteries have associated injury to nerves.

Operation on the axillary or brachial artery was performed in this hospital on 11 patients. Ligation of the artery was performed in 9 cases, and endoaneurysmorrhaphy was done in 2. In 1 of the cases of endoaneurysmorrhaphy the radial pulse persisted normally postoperatively; in the other it did not. Normal circulation in the hands was preserved in both. Of the patients having ligature of the artery, in some a weak palpable radial pulse was preserved, in some the pulse disappeared and in others there was no pulse prior to operation. The level of the ligation (i. e., above or below the profunda brachii artery) apparently had no bearing on this.

In the technic of operation on these two arteries, there are several points that are worth emphasizing. Exposure is, as in all arteries, the most important. The pectoralis major and minor should be divided in any patient who has a wound to the artery that is higher than midarm. This is particularly true if there is an aneurysm or a pulsating hematoma and certainly will have to be done if there is a divided nerve in this region that will need suturing. In a patient with aneurysm or pulsating hematoma whose wound is so proximal that a tourniquet cannot be applied above it, the preliminary dissection should be most careful and prolonged. Two or three hours may be necessary in order to get well behind the lesion and around it on as many sides as possible before it is broken into. This is important because the hemorrhage caused by breaking into the aneurysm can always be easily controlled if the operator is in a position to lift the whole neurovascular bundle forward with the fingers, thereby applying enough tension to stop bleeding and allow careful dissection of the lesion (fig. 2). In all cases a provisional ligature of tape tied down over a rubber tube in order to protect the wall of the vessel from the trauma of the knot [4] is placed on the main artery at the nearest spot to the aneurysm that is easily

4. Watson, G., cited by Makins.[1]

accessible. One of the most difficult features to deal with in dissecting out lesions of the brachial and axillary arteries is hemorrhage from the venae comitantes and their branches, which communicate across in front of the artery. These are often involved in scar and have thin walls. If they cannot always be dealt with by ordinary dissection, they can be secured with the neurovascular bundle on tension as described in the dissection of the artery.

The great majority of our patients were received with limbs in plaster casts. This included some patients who had no fractures and who had been immobilized presumably to protect their large wounds according to the teachings of Trueta.[5] Two of these were definitely felt to have been harmed by this procedure, 1 having an amputation

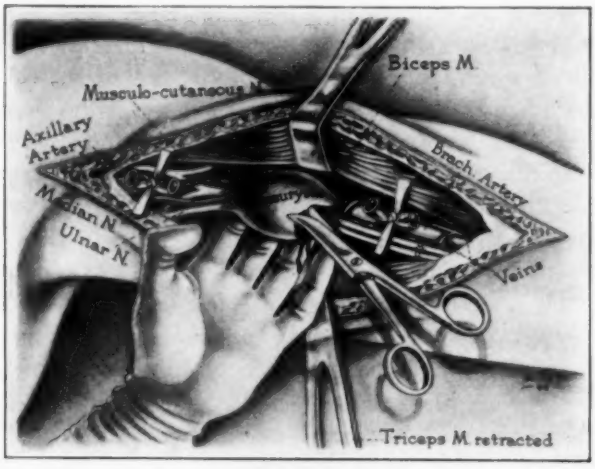

Fig. 2.—Diagrammatic representation of the technic of approach to an aneurysm of the brachial artery. Note Watson's temporary ligatures in place on the artery.

through the forearm at a higher level than had been expected before the limb had been put in plaster and the other receiving a stiff and edematous hand, with some areas of pressure necrosis beneath the cast. It is our conviction that, regardless of fractures, no patient who has had ligature of a main artery of a limb should be placed in a circular bandage of any kind, least of all a plaster cast, until the circulation of the limb has become stabilized at the level which it is finally going to attain. This means that if the patient has no gangrene he shall be kept free for a period sufficient to insure that no gangrene will ensue (at least three days) and that if he does have gangrene he be kept free either

5. Trueta, J.: Treatment of War Wounds and Fractures with Special Reference to the Closed Method as Used in the War in Spain, New York, Paul B. Hoeber, Inc., 1940.

until the gangrene is completely dry, with no surrounding edema, or until amputation is done.

Varying degrees of causalgia were frequent in this group. It is difficult to recall a patient with aneurysm or pulsating hematoma that did not suffer this symptom. This could be relieved by dorsal sympathetic block for short periods or, as in the case with the subclavian aneurysm, by dorsal sympathectomy, but the excision of the aneurysm and lysis or suture of the involved nerves were most successful.

The great importance of physical therapy in the treatment of these wounds of the arm has been impressed on us. If there has been any ischemic myositis or paralysis, much can be gained by active and passive motion if they are started early. The presence of dry gangrene is no contraindication to this. In many cases nerves that were thought to be completely paralyzed recovered function in a surprising manner after they had responded to this treatment.

IV. Femoral Artery.—There were 18 patients. Eight of them arrived with amputation already performed, and because of inadequate records as to the level of the arterial injury the cases are not of much value for study. Three patients had amputation below the knee, and the other 5 had amputation above. Of the 10 who arrived without amputation, 6 had had the continuity of the femoral artery interrupted prior to admission. Of these, 2 had gangrenous legs and had amputation at this hospital; 4 had limbs with adequate circulation. The remaining 4, who arrived without amputation, were patients who suffered injury to their femoral arteries without interruption of arterial flow through the area. Two of these had aneurysms which were not operated on in our hospital. A third had an enlarging hematoma requiring ligation of the artery, a lumbar sympathectomy being performed four hours later. The case of the fourth patient was a puzzling and tragic one. Ligation of the profunda femoris artery was necessary because of secondary hemorrhage from severe wounds of the thigh. Further secondary hemorrhage then resulted from the main trunk of the femoral artery which had been seen to be intact at the original operation. It was necessary to ligate the femoral artery in the midst of infection, and the leg was lost, the patient dying subsequently of overwhelming infection due to Klebsiella pneumoniae. The case was of interest in the light of a similar one at another hospital, in which rupture of a previously intact common femoral artery occurred a few days following ligation of the femoral vein. Why a normal main femoral artery should become involved in the process and bleed in both instances has not yet been explained.

Of the four amputations for femoral artery injury performed in this hospital, two were done below the knee and two above. The two

that were done above were extremely high, a subtrochanteric and a hip joint disarticulation.

The related experience has given us a great respect for the common femoral artery. It must be borne in mind, however, that all the ligations performed were in nonelective cases. We have no data on the incidence of gangrene following elective ligation.

V. Profunda Femoris Artery.—There were four lesions of the profunda femoris artery and its branches. One was an arteriovenous aneurysm arising within 1 cm. of the origin of the profunda from the common femoral artery and causing an aneurysmal sac of a dumbbell shape involving the profunda femoris vein and the superficial circumflex iliac vein. This was excised so close to the common femoral artery that a lateral suture of it had to be performed. The other three were secondary hemorrhages from the profunda femoris or its branches in association with subtrochanteric fractures of the neck of the femur. One ligation was done elsewhere; the other two were done here. These patients presented special problems because it was necessary to find the bleeding point, amidst a large amount of callus and damaged muscle, usually after the patient had stopped bleeding. In both patients this took several attempts, with the inevitable result that infection and osteomyelitis ensued. In 1 case this was not serious, but in the other, the 1 in which the common femoral artery gave away after the ligation of the profunda, it meant loss of the leg and, later, loss of the patient's life.

The lesson to be learned from these cases is that when one is presented with secondary arterial hemorrhage from the region of a subtrochanteric fracture of the femur one should prepare for an extensive operation, ligate the profunda femoris artery above the origin of the femoral circumflex branches and then open the whole region of the fracture and secure the bleeding point for sure at the first attempt. The common femoral artery should be disturbed as little as possible during this procedure.

VI. Popliteal Artery.—There were 30 patients with injuries to the popliteal artery. Of these, 22 had gangrene of such an extent that in all but 1 amputation was performed. Four others had atrophy, Volkmann's contracture or ischemic myositis, whatever term one wishes to use. Ten of the 21 who came to amputation had no associated fracture; the other 11 did.

Of the 3 patients who underwent arterial operation at this hospital, gangrene developed in 2 and required later amputation. One of these, a patient who had a near traumatic amputation of his leg in an automobile accident, was received shortly after his accident. His collateral circulation had been completely destroyed by the trauma. A venous graft was attempted but was given up because of technical difficulties arising from

severe damage to the artery over a long area. Amputation was done forty-eight hours later. The other was a battle casualty that had a pulsating hematoma of the popliteal space from a wound of the artery two and a half weeks previously. The hematoma increased in size and eventually dissected almost up to the femoral triangle. Surgical intervention became necessary. There was no back bleeding from the artery at the time of ligation, and during the four hours following it it became obvious that the leg was not going to survive. The wound was therefore reopened, a venous graft sewed in place and the patient immediately heparinized. Although the graft was functioning at the close of procedure and heparinization was continued, the leg became gangrenous and was amputated. Later examination of the artery showed that the graft had become solidly clotted. The third patient, the 1 who did not get gangrene, underwent excision of a popliteal aneurysm one week after lumbar sympathectomy. He did well. There was no question about the viability of his foot at any time following operation.

Supracondylar fractures of the femur with contusion or rupture of the popliteal artery present a special problem because of the severe damage to collateral vessels as well as to the main artery. Of 4 such cases in our series, amputation was done in all and the circulatory damage was so severe that in only 1 of the patients could the knee joint be preserved. In that patient the stump left was only 5 cm. long, measuring from the tibial tubercle.

Of the twenty-one amputations for injury to the popliteal artery, eleven were performed above the knee joint and ten below. In these ten, six of the stumps were so short as not to be of much value for a prosthesis. Of the 4 patients having good length in their below knee stumps 2 had had lesions which had not entailed rupture of the artery but rather thrombosis or spasm. The other 2 had suffered rupture of the artery with hemorrhage into the popliteal space but had had extensive surgical incisions made through the fascia, enabling evacuation of the clot. The patients who underwent amputation close below the knee or above it did not have extensive incisions of this nature. It appears that these incisions to relieve pressure on collateral vessels are decidedly advantageous. Closed as well as open fractures of the lower femur or upper tibia and dislocations of the knee joint which have any evidence of damage to the popliteral artery should have exploratory incisions made in the popliteal space and the incisions left open if there is any evidence of hematoma or swelling.

B. SECONDARY HEMORRHAGE

There were 13 cases in which secondary hemorrhage was severe enough to require operative intervention or to cause death. There were

many other cases of minor hemorrhage which required only packing and of which no records were kept. There were over 9,500 casualties treated during this time. The figure of 13 was far lower than one would have predicted out of this total number.

The ligations that were done for hemorrhage were of the following arteries: common femoral, one; superficial femoral, one; deep femoral, two; popliteal, one; posterior tibial, one; brachial, one; external carotid and common carotid combined, one; external carotid alone, two; internal pudendal, one, and right lumbar, one.

The patients who suffered loss of a leg following treatment of secondary hemorrhage from the popliteal, deep femoral and common femoral arteries have been mentioned. Other interesting special problems and situations were encountered as enumerated in the following paragraphs.

One patient who had a severe secondary hemorrhage from a wound which perforated the cecum and injured a right lumbar vessel required a diversion of the intestinal stream from the area by an ileotransverse colostomy before the bleeding was satisfactorily controlled.

One patient who had suffered a gunshot wound of the neck died suddenly from rupture of a traumatic aneurysm of the superior thyroid artery into the larynx, a lesion which had previously been in no way suspected.

One patient had twelve secondary hemorrhages from his mouth following a penetrating oblique wound from a shell traversing the submental region, shattering his right mandible and causing aphasia and right hemiplegia from contusion of the left internal carotid artery. Although three of these hemorrhages were so severe as to result in almost complete exsanguination, their source, which turned out to be a branch of the left lingual artery presenting in the pharynx 2 cm. posterior to the fauces, was not found until the pharynx was examined with the patient under general anesthesia. Prior to this the opinion was that the bleeding was coming from the shattered right mandible, since complete visualization of the posterior pharynx was not possible, due to trismus.

Another patient suffered a gushing hemorrhage from a small aneurysm of the internal maxillary artery presenting below the inferior turbinate in the left naris. This would have been rapidly fatal had not the Chief of the Section of Otorhinolaryngology, Capt. A. C. Johnson, been in the ward at the time of the occurrence to insert a packing. He ligated the external carotid artery immediately and later injected sclerosing solution into the area. Many weeks later the aneurysm was seen to be in a much collapsed state.

In general our impressions with regard to secondary hemorrhage are that: (*a*) if a main artery of a limb must be ligated as an emergency

measure for hemorrhage the limb stands a much poorer chance of survival than if the ligation can be done as an elective procedure; (*b*) hemorrhages from traumatic lesions of arteries lying in or beneath mucous membranes are particularly perilous, and (*c*) if the hemorrhage is going to be attacked surgically this should be done radically and completely at the first operation.

C. COMMENT

I. The critical arteries are the popliteal, the internal carotid and the common femoral. Others become critical if the collateral circulation has been injured by the wound. This must always be taken into account when a case is evaluated.

II. The place of sympathectomy or repeated anesthesia of the sympathetic chain is not easy to evaluate. It is our definite impression, however, that the chief time for sympathectomy is prior to operative interruption of a critical artery. Its use as a prophylactic against spasm of collateral arterial supply is its most important function. If there is not time to do it before and it can be performed within six hours after such interruption, it is also worth doing but much less benefit can be expected therefrom. The one exception to this occurs in the event that the arterial lesion at the time of the débridement has been shown to be one of spasm, as occurred in one injury to the popliteal artery that we observed subsequent to this group. Sympathectomy is then the treatment of choice at any time.

Procaine hydrochloride injected paravertebrally should be used in cases of arterial wounds only in which the condition of the patient does not warrant the operation of sympathectomy, and the injections should be repeated intermittently as frequently as practicable for as long as a favorable response is obtained. This usually will not be longer than forty-eight hours after injury.

These recommendations sound radical, but they in fact advocate meddling with the sympathetic systems of fewer patients than has been the general custom. The patient in whom procaine hydrochloride injected paravertebrally is to our mind of little value is the familiar one who is seen a day or two after injury and in whom ischemic demarcation has already started. We have observed little benefit either from the standpoint of saving the limb or from that of lowering the level of demarcation by the use of procaine hydrochloride injected paravertebrally or of sympathectomy in these cases.

III. The pattern of gangrene in limbs with injuries to critical arteries is a subject on which much interesting work is still to be done. The fact that in all such patients the skin is viable well below the level of dead muscle is a common observation. Extreme examples of this

have occurred when 2 patients with injury to the popliteal and brachial arteries respectively presented external evidence of gangrene of only the tips of the fingers and toes. As time passed, liquefaction of all the muscles of the calf in 1 case and those of the forarm in the other supervened, without further cutaneous gangrene.

The interesting anatomic observations of Clark and Blomfield,[6] which divide muscles into various groups based on the pattern of the arterial circulation within them, demonstrate that the gastrocnemius and soleus muscles possess a far poorer potential collateral circulation than the deeper muscles of the leg. It has in general been our experience that these two muscles show gangrene to a higher level than the deeper ones. The importance of doing selective débridement of these muscles at the time of amputation is evident.

IV. The importance of a test or sign which would enable surgeons to tell preoperatively whether ligation of a given artery will result in gangrene is obvious. The practical importance of such a test would be that it would tell whether it is necessary to attempt an anastomosis or venous graft or whether simple ligation is safe. We have used the Henle-Coenen test.[7] This consists in consideration of the briskness of the blood flow from the distal cut end of the artery as observed on the operating table as an indication of the amount of arterial pressure and blood flow being brought into the distal limb by the collateral vessels. In no case in which we have ligated an artery using this sign as an indication has any trouble ensued. In 1 case the complete lack of back bleeding from the distal cut stump of a popliteal artery prompted us to sew in a venous graft. The graft unfortunately failed, and gangrene ensued, showing that one's fears based solely on this sign were well founded. Our experience with emergency ligations has been limited, however, and personal communications from surgeons in forward hospitals who have been intimately associated with the emergency cases say that this sign has been unreliable. The state of the vessels of the limb is at that time in a transitory stage. The amount of spasm and thrombosis of the collateral vessels which will eventually be present has not yet become established. This consideration apparently may invalidate any early decision that is made. For the late elective case, however, in which these factors are stabilized, the sign in the opinion of my colleagues and me is a valuable one.

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6. Clark, W. E. L., and Blomfield, L. B.: The Efficiency of Intramuscular Anastomosis with Observations on the Regeneration of Devascularized Muscle, J. Anat. **79**:15 (Jan.) 1945.

7. Matas, R.: Testing the Efficiency of the Collateral Circulation as a Preliminary to the Occlusion of the Great Surgical Arteries, J. A. M. A. **68**: 1441 (Oct. 24) 1914.

SUMMARY

The following are the most important lessons and impressions that have been learned from the experiences related.

1. The critical arteries are the popliteal, the internal carotid and the common femoral. Others become critical if the collateral circulation has been involved in the wound.

2. Early ligation or ligation for secondary hemorrhage is dangerous. Late or elective ligation is much less so.

3. Sympathectomy should be done if possible before or immediately after ligation of critical arteries. If impossible, sympathetic blocks should be used for forty-eight hours.

4. The release from closed spaces, such as the popliteal space, of blood clot or edema through large incisions which are left open is of extreme importance in the affording of free flow through collateral vessels. The particular application of this procedure to patients with supracondylar fractures of the femur is emphasized.

5. In early cases of wounds of critical arteries the use of venous grafts or prostheses is indicated.

6. In the decision as to whether a graft or prosthesis should be used in a patient having a late or elective ligation, lack of good arterial back bleeding from the distal stump is the one most important indication for their use.

7. In operations on hematomas or aneurysms, long incisions, wide dissection (preserving as much collateral circulation as possible in the process) and provisional ligatures with tape over a rubber tube are recommended.

8. A circular bandage should not be used in any limb that has doubtful circulation. Patients with such limbs should not be moved from the hospital where they were first admitted until the circulation is stabilized.

Major Charles L. Neill and Capt. Ludwig H. Segerberg, of the Section of Neurosurgery, allowed me to include the neurologic cases in this series.

GLOMUS TUMORS (ANGIONEUROMYOMAS)

A Clinical and Pathologic Report of an Unusual Case

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AND
RUTH BALKIN, M.D.
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IT HAS long been taught that the dilatation and contraction of the capillaries of the skin helps to regulate the heat of the body. That there is an accessory vascular apparatus called the glomus also present is not so generally appreciated.

HISTORICAL DATA

In 1920, Barré, a French surgeon, removed a tiny painful tumor from beneath a finger nail and gave it to P. Masson for pathologic study. In his report of this tumor in 1924 [1] Masson gave to the world the first description of a neoplasm originating in a glomus. It was bluish red and 3 to 4 mm. in diameter and was composed of large, clear, round or polyhedral cells, which were similar to those found in the coccygeal gland or glomus. Besides these "epithelioid" cells, there were present in the tumor mass arterioles, veins, smooth muscle cells and nerve fibers. Masson was so struck by this conglomeration of epithelioid cells, blood vessels, nerves and muscle that he combed the anatomic atlases for a possible explanation. He found that Sucquet [2] in 1862 first described peculiar arterioles in the palms and soles which opened directly into neighboring veins, without forming a capillary network. Further search revealed that H. Hoyer [3] had given a more detailed description of an arteriovenous anastomosis in the skin of adults. This anastomosis takes the form of a coiled arteriovenous mechanism, which Masson named the Sucquet-Hoyer canal. Masson was forced to the conclusion that Barré's tumor was an organoid overgrowth of all the elements that go to make up the glomus entity. He thus was the first investigator to describe this tumor and to explain the anatomic and physiologic implications of it.

Read before the Chicago Surgical Society Dec. 7, 1945.

1. Masson, P.: Le glomus neuromyo-artériel des regions tactiles et ses tumeurs, Lyon chir. **21**:257 (May-June) 1924.

2. Sucquet, cited by Testut, J. L.: Traite d'anatomie humaine, Paris, O. Doin, 1921, vol. 2, p. 101.

3. Hoyer, H.: Ueber unmittelbare Einmündung kleinster Arterien in Gefäss-äte venösen Characters, Arch. f. mikr. Anat. **13**:603, 1877.

Ten years later, Nicholas W. Popoff,[4] of Rochester, N. Y., published a paper entitled "The Digital Vascular System." Popoff had been impressed by the increasing number of painful tumors of the skin of the extremities that had been reported since Masson's report in 1924, and he decided to make a detailed anatomic study of the skin of the digits. He was rewarded in his search by the finding of large numbers of a normal anatomic entity, the glomus, consisting of an afferent artery which breaks into a coiled arterial organ—the Sucquet-Hoyer canal. Furthermore, he found preglomic arterioles, with clear periglomic expansion zones, furnished with neuroreticular mechanisms of sympathetic and myelinated nerves, which control the Sucquet-Hoyer canal. The glomus also has a specially arranged system of collecting veins and an outer lamellated collagenous zone surrounding the entire glomus. By this mechanism, a large amount of blood can quickly be shunted into the skin to help regulate the general or local temperature of the body. Popoff found most of these glomus units on the digits and in the palms and soles. Large numbers, however, were also present in the skin of the extremities and lesser numbers on the trunks of adults. He found none in infants, a fact which explains their inability to accommodate themselves to sudden changes of temperature. Glomus units have been found in all warm-blooded animals.

The first glomus tumor to be reported in this country was seen by Mason and Weil [5] in 1934. Since that time, some three hundred glomus tumors have been collected from the literature. Before they were classified as glomus, the older clinicians knew them as painful cutaneous tubercles. O. T. Bailey,[6] writing in "Oxford Medicine" in 1939, collected 100 cases and gave the tumor the title of glomangioma. The *Quarterly Cumulative Index Medicus* of the American Medical Association classes them as angioneuromyomas. Beaton and Davis reported 3 cases in 1941 and collected 271 cases from the literature.[7]

ETIOLOGY

There is no known etiologic factor; trauma has not been found to be a cause. The two sexes are attacked equally, and no race is immune.

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4. Popoff, N. W.: Digital Vascular System, with Reference to State of Glomus in Inflammation, Arteriosclerotic Gangrene, Diabetic Gangrene, Thromboangiitis Obliterans and Supernumerary Digits in Man, Arch. Path. **18**:295 (Sept.) 1934.

5. Mason, M. L., and Weil, A.: Tumor of Subcutaneous Glomus, Surg., Gynec. & Obst. **58**:807 (May) 1934.

6. Bailey, O. T., in Christian, H. A.: Oxford Medicine, New York, Oxford University Press, 1939.

7. Beaton, L. E., and Davis, L.: Glomus Tumor: Report of Three Cases, Quart. Bull., Northwestern Univ. M. School **15**:245, 1941.

SYMPTOMS

The tumors are usually small, ranging from 2 to 5 mm. in diameter. Some have reached 3 cm. in diameter. They are of firm consistency and limited growth. They are usually blue or red, the degree of color depending on the size or depth of the tumor. They are well circumscribed and movable when in the cutaneous tissues, where they are superficial to the superficial fascia. Many are found beneath the nails of the fingers or toes, where they produce excruciating pain, which often radiates up the arm to the face or along the leg for long distances. The pain is often lancinating or stabbing or pulsing in character and is activated by contact with the outer clothing or changes in temperature, or it may occur spontaneously. Pulsative tumors may erode bone when in contact. Atrophy of muscle may be associated but is usually a phenomenon of disuse of the part due to the pain. When the tumor is subungual, the nail may be curved and thickened, with longitudinal striations. The nails are often left uncut because of the severe pain. When excised completely, the lesions usually do not recur. Pain usually ceases after removal of the tumor, but sometimes it persists for a time. The tumor usually occurs singly, but it may be multiple; as many as forty-eight have been found in 1 person. Two-thirds of the reported tumors have been located on the upper extremities.

The tumors appear early in middle life and may be present for several years before the patient seeks relief of the pain or discomfort.

DIAGNOSIS

The firm, circumscribed, superficial nodules, bluish or red, the presence of pain out of all proportion to the size of the tumor and the demonstration by microscopic section of the presence of layers of large epithelioid glomus cells with myelin and nonmyelin nerve fibers in direct continuity with the cytoplasm are pathognomonic.

DIFFERENTIAL DIAGNOSIS

The true glomus tumor must be differentiated from most benign tumors of the skin such as fibroma, angioma and angiomatous leiomyoma. The malignant sarcomas, melanoblastomas and carcinomas are easily differentiated by their more rapid growth and microscopic characteristics. Felon, subungual exostosis or periostitis and subungual wart may produce pain.

TREATMENT

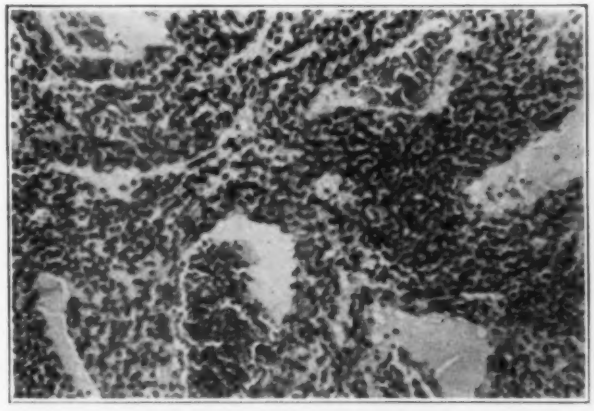
Complete excision usually cures. In the nail bed, the nail must be excised over the tumor to allow a complete excision. In the skin the mass is easily excised from the surrounding subcutaneous tissue, with the patient under local anesthesia.

REPORT OF A CASE

A Negro coachman and houseman, 68 years old, first noticed a small nodule in the skin of the flexor surface of the left forearm, just below the elbow, twenty-five years before he consulted us. It grew slowly and did not cause appreciable pain until about five years later. For the past two years the pain, beating in character, has become more insistent and has awakened him from sleep. For the past year it has interfered with the use of the arm.

On examination, the mass is slightly movable over the brachioradialis muscle, in which it has caused a depression. The skin is attached but not discolored. The mass is about 4 cm. in diameter and slightly sensitive to manipulation. The largest tumor of this type heretofore reported has measured 3 cm.

Results of the general examination of the patient were negative as was also the reaction to the Wassermann test. Past history is noncontributory except for this new growth. The patient asks that the tumor be removed on account of the pain, spontaneous and throbbing but not radiating, which has now become unbearable.



Photomicrograph showing layers of epithelioid cells and dilated vascular channels.

Treatment.—With the patient under local anesthesia the mass was excised from the surrounding fatty subcutaneous tissue, and healing took place uneventfully.

Pathology.—The specimen consists of a well encapsulated tumor that measures 4 by 2.5 by 2 cm. It is moderately firm, and the sectioned surface is deep red and has a spongy appearance.

Microscopic section of the tumor reveals it to be encased in a dense hyaline connective tissue capsule. The tumor itself is composed of numerous vascular channels which are the size of dilated capillaries, such as are seen in capillary hemangiomas. These channels have walls that consist of a single layer of endothelium and are surrounded by cords and sheets of cells which are round to oval in shape and regular in size and have poorly defined cell membranes. In some areas these cells are rather closely packed together, but in other zones they are more widely spaced. However, in both areas an eosinophilic fibrillar meshwork can be seen between the cells. Occasional short, thin strands of smooth muscle can be seen in the tumor as well as small thick-walled arteries, which are most numerous at the periphery.

DISCUSSION

DR. HILLIER DAVIS: A patient operated on by me at the Cook County Hospital several years ago showed a mass in the omentum and multiple tumors of the intestine, which bled readily when touched.

DR. RUTH BALKIN: Dr. R. Jaffe performed an autopsy on this patient and found a primary tumor of the omentum, with metastases throughout the abdomen. The tumor had all the characteristics of a glomus, and in his opinion this was a unique case, as no malignant growth of this type had ever been reported up to that time and there have been none reported since.

MIGRATORY POLYPHLEBITIS

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MOUNTING interest in peripheral vascular diseases is evident from the large volume of literature on this subject in textbooks and current surgical journals. However, one type of structural vascular disease infrequently discussed is migrating phlebitis.

This type of phlebitis is a primary inflammatory lesion of a venous segment with secondary vasomotor disturbances. Characteristically, it involves a segment of vein and subsides in the original area only to appear in another segment of vein in the same or opposite leg. Because of this tendency it is my preference to term this entity migratory polyphlebitis.

Polyphlebitis of this type usually involves the surface veins of the legs. However, it may occur in the deeper veins. The upper extremities may become the site of this disease as well as the lower extremities. I have seen vessels of the anterior part of the abdominal wall involved with this process.

ETIOLOGY

Migratory polyphlebitis when not associated with thromboangiitis obliterans (Buerger's disease) should be considered as a forerunner of this disease. Thus the etiology falls into the same category as that of thromboangiitis obliterans. However, migratory polyphlebitis is not confined to the Jewish race, nor does it have a high incidence in this race, as does thromboangiitis obliterans. The 4 patients under present discussion were all male patients, none of whom were Jewish. Migratory polyphlebitis occurs almost always in men. This fact has led some investigators to state the belief that estrogenic hormones are beneficial as a therapeutic measure in the treatment of this disease.[1] Usually the disease is seen in young adults, between 20 and 40 years of age. Some authors have expressed the opinion that there is a familial trait or tendency toward thromboangiitis obliterans, and this may also be applied to migratory polyphlebitis.[2] In view of the inflammatory reaction of this disease process, many investigators have cultured

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From the surgical service of Dr. John F. Raycroft, Kings County Hospital.

1. McGrath, E. J.: Experimental Peripheral Gangrene, J. A. M. A. **105**:854 (Sept. 14) 1935.

2. Collens, W. S., and Wilensky, N. D.: Peripheral Vascular Diseases, Springfield, Ill., Charles C Thomas, Publisher, 1939.

various types of bacteria.[3] The role of tobacco has been accentuated as being deleterious to all types of patients with peripheral vascular diseases. The basis for this is the knowledge of the profound spasm of peripheral vessels which occurs in the presence of nicotine. However, it is doubted that there is a causal etiologic relationship between tobacco and inflammatory lesions of blood vessels. Once the disease has occurred, tobacco should be considered as definitely deleterious.

In a discussion of the etiology of this disease, mention should be made of the fact that in my experience almost all patients with migratory polyphlebitis present a significant antecedent history. The historical data may be those of trauma to the extremities, frostbite or an acute systemic febrile disease such as pneumonia. Although these factors do not precipitate an acute episode of polyphlebitis, they may be initiating factors in the production of the disease in a patient with an underlying tendency toward peripheral vascular impairment.

PATHOLOGY

Migratory polyphlebitis is an inflammatory lesion. It is a primary structural vascular disease which may cause secondary vasomotor disturbances. This disease is characterized by the fact that it involves venous segments and recedes in the original area only to commence in a segment of vein elsewhere. Its migratory tendency is similar to that of migratory polyarthritis in acute rheumatic fever. This is significant because a segmental occlusion of a major vessel does not necessarily interfere with the collateral circulatory function.

During the acute phase of polyphlebitis the superficial veins are affected. The involved segment is acutely inflamed, reddened and tender to palpation. The condition in an involved segment will run a course from seven to fourteen days. Subsidence of the acute infection results in a hard vein similar to a goose quill. Pigmentation occurs about the segment. With the onset of pigmentation, another venous segment may become involved. Early lesions are confined to veins alone. However, as the disease progresses, the arteries may become involved. Careful interrogation of patients with full-blown thromboangiitis obliterans will often elicit a history of migratory polyphlebitis. It may, therefore, be considered a forerunner or a manifestation of thromboangiitis obliterans.

CLINICAL PICTURE

The patient is usually a young man. The significant physical findings are revealed in the area of complaint. This is usually a venous segment of the leg which presents the physical characteristics previously

3. Allen, E. V., and Lauderdale, T. L.: Accidental Transmission of Thromboangiitis Obliterans from Man to Man, Proc. Staff Meet., Mayo Clin. **11**:641, 1936. Goodman, C.: A Skin Test to Suggest the Diagnosis of Typhus and Thromboangiitis Obliterans, Bull. New York Acad. Med. **11**:403, 1935.

mentioned. The lesion is fundamentally an acute inflammatory process without systemic response to an inflammatory reaction such as chills or fever. When migratory polyphlebitis involves the deeper veins, the diagnosis is most difficult, since there is no redness, tenderness or other evidence of acute inflammation. However, deep vascular involvement in the leg may produce a positive Homan sign, cramps or edema resulting from venous obstruction. Edema of this type is identified by the rapidity with which it appears.

The following cases have come under my personal observation during the past three years. Each presents a most interesting lesson in migratory polyphlebitis.

Case 1.—The patient, Frank W., aged 24, was seen Dec. 1, 1943, at which time he presented a history of having had pleurisy and pneumonia in 1940. One year later he noticed reddened areas over the veins of his legs, which would last about two weeks and disappear only to appear elsewhere. This process continued intermittently for about a year. He visited his doctor, who advised and performed a bilateral ligation high in the saphenous vein. Shortly thereafter slight edema of both legs developed. In December 1943 he entered Kings County Hospital because of the presence of reddened veins in the anterior part of the abdominal wall which were similar to those which had developed previously in his legs. A biopsy was performed, a portion of the vessels of the anterior part of the abdominal wall being excised, and was reported to show thromboangiitis obliterans. The patient was treated conservatively. He was sent to a home for convalescents in the upper part of New York state. On March 10, 1944 signs of an acute condition within the abdomen developed. He was operated on, and the following significant conditions were unfolded: (1) superior mesenteric thrombosis, (2) thrombosis of the portal and hepatic veins and (3) acute gangrenous appendicitis with thrombosis of the appendical vessels. The patient died shortly after operation.

Case 2.—The patient, William T., aged 19, was seen in consultation on Jan. 21, 1946. The history is one of fifteen months' duration. His first complaint occurred at the time when reddened, indurated areas developed about his left ankle, which disappeared only to appear about his left knee several weeks later. After this episode, he had a similar experience with the development of reddened areas above the left knee. During these fifteen months he had sporadic episodes of edema of the left ankle. Three days prior to his present examination he noticed two reddened areas on the medial aspect of his right tibia. He had no systemic reaction such as chills or fever during any part of the fifteen month period. Examination was essentially noncontributory excepting for the pertinent findings in the lower extremities. Both legs were grossly symmetric in size, shape and development. Bilateral pulsations of the major vessels were demonstrated. There was no alteration of the temperature of either extremity. There was no edema. Several segmental surface veins about the medial malleolus of the left leg were tender and red and indicated inflammatory changes. In the midtibial region of the medial aspect of the right leg there was a segment of vein approximately 5 cm. in length which was red, tense and moderately tender to palpation. The patient was given conservative therapy and responded to the prophylactic therapeutic regimen. This case demonstrates the early process of migratory polyphlebitis.

CASE 3.—The patient, James C., aged 39, was admitted to Kings County Hospital Feb. 1, 1946. He presented a history of having had frostbite twenty years before. Five years later he had trouble with his veins, the nature of which he did not recall. About five years before I saw him he noticed hardened, reddened areas about his legs, which were tender to touch. These areas would disappear for a period of several months and then would occur in the opposite leg. This caused him no distress or discomfort except for the fact that nocturnal edema developed on several occasions. He entered the hospital at this time because of severe pain in both legs. At this time he has a classic picture of thromboangiitis obliterans. This patient demonstrates a past history of migratory polyphlebitis which was characterized by exacerbations and recrudescences over a period of twenty years. Finally he sought medical care because of characteristic changes subsequent to classic thromboangiitis obliterans.

Fig. 1.—*A*, photograph of the foot of the patient in case 3, illustrating classic thromboangiitis obliterans in a patient with a previous history of migratory polyphlebitis. *B*, photograph of the foot of the patient described in case 4, illustrating the healing phase of migratory polyphlebitis, with desquamation of the skin over the involved vein. Trophic changes in the toes are clearly demonstrable.

CASE 4.—The patient, John F., aged 26, was brought to the hospital after exposure, which produced what was thought to be frostbite of both toes. He entered the hospital on Feb. 4, 1946, with a history of drunkenness following which he slept out-of-doors in the snow, finally being brought to the hospital. Examination revealed significant findings in both lower extremities. There were segmental involved venous radicles in the feet and in the thighs. These revelations indicated typical migratory polyphlebitis. This patient responded to conservative therapy and has progressed to the chronic stage, with pigment formation and the production of goose-quill-like veins.

These cases demonstrate the multivarious aspects of migratory polyphlebitis. The possible complications and the eventual course of the disease are clearly unfolded.

DIFFERENTIAL DIAGNOSIS

The characteristic features of migratory polythrombophlebitis are as follows:

1. The disease involves superficial venous segments in the early stages.

2. Discrete nodules form, varying in size from 2 to 4 mm. to 4 or 5 cm.

3. The adjacent veins may contain thrombi.

4. The lesions appear first on the dorsal areas of the feet, legs and forearms and may involve the vessels of the abdominal wall.

5. The acute phase lasts one or two weeks, the lesion disappearing slowly and appearing elsewhere either singly or in groups.

6. When healing occurs, the veins become tense and scarred.

7. Brownish pigmentation with epithelial desquamation occurs over and above the veins when healing commences.

This pathologic process must be differentiated especially from erythema nodosum when the phlebitis is in the anterior parts of the tibial areas. Other forms of nonspecific thrombophlebitis which may occur in varicosities must be eliminated. Phlebitis occurring as a complication of acute febrile disease, erysipelas and similar lesions must be differentiated from migratory polyphlebitis.

PROGNOSIS AND TREATMENT

As has been previously stated, the appearance of migratory polyphlebitis must be considered as an antecedent to thromboangiitis obliterans. Without adequate treatment, patients with migratory polyphlebitis eventually become candidates for all the complications usually associated with thromboangiitis obliterans. When this occurs the prognosis becomes the same as that of thromboangiitis obliterans. The treatment for this disease entity divides itself into three major groups: (1) prophylactic measures, (2) medical treatment and (3) surgical treatment.

Prophylaxis against phlebitis of this type is of considerable value, especially in postoperative and postpartum patients. Any person presenting a history remotely suggestive of migratory polyphlebitis who must have elective operation not related to his circulatory system should be considered for early ambulation following operation. It has been our policy for all patients after operation to perform motion of the feet as early as possible. The patient is instructed to move the ball of each foot up and down as often as possible. The motion is similar to stepping on the gas of an automobile. One or both feet are moved at the same time. The patient is instructed that these exercises should be done almost a thousand times in twenty-four hours.

Patients who have a past or present history of migratory polyphlebitis are asked to avoid vasoconstricting affects of cold water. They are also instructed most thoroughly in the general care of the feet. These patients must absolutely eliminate smoking.

Conservative medical treatment is a most satisfactory method in early cases of migratory polythrombophlebitis. In this stage of the disease, venous compression at regular intervals is a most valuable therapeutic measure. This procedure relieves pain and accentuates the disappearance of the acute lesions. The use of Ace bandages is a recommended procedure during an acute attack. The patient may be placed at rest in bed, with elevation of both legs and application of dry heat to the areas involved. The use of chemotherapeutic drugs and/or penicillin has been employed.

Surgical intervention is indicated in cases of deep migratory polyphlebitis when there is much pain, tenderness and edema. In these circumstances, lumbar sympathectomy has been employed most efficaciously. There has been no tendency toward pulmonary embolism in the acute stages of migratory polythrombophlebitis. In the event that sublethal pulmonary embolism should occur, venous ligation would be advised. I have not employed lumbar sympathectomy in any of my cases. When the classic picture of thromboangiitis obliterans develops, the patients are subjected to the same regimen as prescribed for the treatment of those with thromboangiitis obliterans. Any complications arising are treated similarly to those arising in thromboangiitis obliterans.

Miss Dorothea E. Chamberlain aided in the preparation of the manuscript, and Mr. John C. Knight aided with the photography.